The Journal of the American Medical Association

Published Under the Auspices of the Board of Trustees

VOL. 117, No. 5 — AUGUST 2, 1941

COPYRIGHT, 1941, BY AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILLINOIS

EXAMINATION OF THE HEART FOR MILITARY SERVICE

CHAIRMAN'S ADDRESS

FRED M. SMITH, M.D.
IOWA CITY

The determination of the fitness of the heart for military service presents problems that are seldom encountered in civil practice. Thus it is frequently necessary for the examiner to rely on signs alone, and these may be of questionable significance. Tachycardia, systolic murmurs and slight elevation in the blood pressure are commonly observed. Certain persons with tachycardia give poor response to exercise. This may have resulted from sedentary life or from chronic infection, or perhaps the history may disclose that it has been present since childhood.

Acceleration of the cardiac rate is often the first and maybe the only feature that attracts the attention of the examining physician. This is usually due to excitement incident to the examination and thus promptly subsides as the subject becomes relaxed. If, however, it persists, a careful search for a cause is demanded. Tachycardia may be caused by various conditions but is more commonly due to infection or irritable heart.

The response of the cardiac rate to a given amount of exercise does not necessarily provide information concerning the efficiency of the heart, nor does it determine the presence or absence of disease. It is of value, however, in estimating the general physical fitness. If the rate becomes excessive and the time required for it to return to the original level exceeds two minutes, the response is not satisfactory. This is often due to poor physical condition from various causes and not to the heart alone. Thus the response of persons with chronic valvular heart disease may be good, whereas that of one who has led a sedentary life or has an infection may be poor. In questionable cases the history with reference to the participation in physical activities and the response provides much better means of estimating the possibilities of the individual, provided he is free from disease. If it is found that he has been capable of withstanding strenuous exercise in the past, there is good possibility that he may qualify for military service.

The work of the cardiovascular boards during the first World War was chiefly concerned with the examination of men presenting varying degrees of irritable heart, commonly designated as neurocirculatory asthenia, or effort syndrome. The reduced tolerance to exercise is the most characteristic feature of this disorder. Thus on slight or moderate exercise the subject experiences shortness of breath, palpitation of the heart and fatigue comparable to that normally produced by the more strenuous and sustained form of physical effort. Others in addition may have various other symptoms, such as precordial distress, giddiness or perhaps headache. All have unstable vasomotor systems, perspire freely and present cyanosis of the hands when the latter are in the dependent position. Some give a history of having had reduced tolerance to exercise since childhood. In another group the condition was associated with various chronic infections, particularly pulmonary tuberculosis, or followed severe acute infection, such as influenzal pneumonia. Finally in a number the disorder developed while engaged in active warfare. In this connection the series of cases studied by Lewis [1] are of particular interest. Lewis found that the majority of his patients were recruited from sedentary or light occupations. Moreover, the history disclosed that a large percentage of this group had the disorder in civil life and that many of these had been forced to adopt sedentary occupations. It is apparent from the results of this analysis that it is commonly possible to discover or suspect this condition before the subject is inducted into military service.

Alterations in the character of the heart sounds are seldom of much help in the diagnosis of heart disease. Accentuation of the first sound at the apex may be a prominent feature in mitral stenosis. However, it is generally associated with presystolic murmur. In the early stages of mitral stenosis the murmur is frequently confined to a small area and may not be elicited except immediately following exercise, particularly with the subject in the recumbent position and turned toward the left side. Intensification of the second pulmonic sound is present in mitral stenosis, and a loud aortic second sound results from hypertension. Alteration in the character of the latter, especially tympanitic quality, is occasionally the first feature to suggest syphilitic aortitis and the possibility of a diastolic murmur. This murmur may escape detection unless a careful search is made. It is more easily heard by direct auscultation when the subject is in the sitting posture and while he is holding his breath after complete exhalation.

Systolic murmurs over the apex and the pulmonary area are common in men of the draft age, especially in those with tachycardia and slight elevation in the blood pressure. These murmurs are usually soft blowing in character, seldom transmitted to any significant extent, and frequently are altered by posture and respiration. Those of the cardiopulmonary variety are loudest at the end of inspiration and may entirely disappear at the end of forced expiration. There are many instances in which a final decision is dependent on additional information. This is especially true if the murmur is rather prominent and is constantly heard when the subject is in the standing and recumbent positions. A harsh mur-

Read before the Section on Practice of Medicine at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 5, 1941.

1. Lewis, Thomas: The Soldier's Heart and the Effort Syndrome, New York, Paul B. Hoeber, 1920.

mur at the apex is invariably indicative of structural alterations in the mitral valve and it is generally possible to demonstrate a presystolic murmur. In many instances, however, the history is negative for rheumatic fever. Levine [2] has emphasized the importance of grading murmurs with reference to their intensity and states that those of grade 2 intensity and above are generally due to organic disease. He further points out that a systolic murmur has greater significance if the heart rate is slow, the blood pressure is low and there is no anemia. It is, of course, well to bear in mind that a faint systolic murmur at the apex may be on an organic basis; consequently the correct evaluation of this sign is of great importance. The history of rheumatic fever, a diastolic murmur or increase in the size of the heart settles the issue.

A harsh murmur with the greatest intensity over the pulmonic area in the third and fourth left interspace, particularly when accompanied by a thrill, is generally due to one or more congenital anomalies. Further confirmation is obtained by the size and shape of the heart and by the electrocardiogram, if there is a conspicuous right axis deviation. The history is usually negative for rheumatic fever but may disclose that a murmur has been present since infancy.

The size of the heart is often the deciding factor in determining the presence or absence of heart disease. Ordinarily it is possible to locate, with a fair degree of accuracy, the left border of the heart by percussion.[3] This is best accomplished with the subject in the sitting position and by direct percussion. If there is doubt regarding the results, they may be checked by the location of the apex impulse. In the normal subject the apex impulse is limited to a relatively small area and usually located in the fifth interspace, well within the nipple line. The outermost point at which the examining finger is raised by the heart represents the left border at this level and generally checks with the results obtained by percussion. The location of the apex beat varies with the type of chest. Thus in those with short thick chests it is at a high level and may extend to the nipple line. In general, however, if the apex impulse extends beyond this point it is indicative of cardiac enlargement. An unduly prominent apex beat or one occupying a relatively large area is ordinarily associated with enlargement of the heart. In persons with a thin chest and a hyperactive heart the impulse may be rather diffuse. This, however, disappears with the recession of the cardiac rate.

Frequently the blood pressure is slightly elevated with tachycardia but usually returns to the normal with the disappearance of the latter. If under these circumstances and after repeated determinations the systolic pressure is 150 or more and diastolic is 90 or more mm. of mercury it is indicative of hypertension. Further investigation may demonstrate the presence of albumin and casts in the urine or, in rare instances, coarctation of the aorta. In the vast majority of cases no cause is found and the condition is listed as essential hypertension.

Special laboratory procedures, such as the use of the roentgen ray and the electrocardiogram, are seldom necessary in this type of examination. Occasionally a roentgenogram is advisable to check the size and shape of the heart. In rare instances it may be difficult to differentiate between premature beats and auricular fibrillation by auscultation, and thus an electrocardiogram is indicated. Auricular fibrillation, however, is generally associated with obvious structural changes in the heart. Sinus tachycardia may suggest paroxysmal tachycardia or perhaps auricular flutter. The fact that the cardiac rate in the former subsides with relaxation dispels any doubt about the situation. Finally the taking of an electrocardiogram may be justified, if the heart rate is unusually slow, in order to rule out the possibility of auriculoventricular block.

2. Levine, S. A.: The Systolic Murmur: Its Clinical Significance, J. A. M. A. **101**:436 (Aug. 5) 1933.

3. Mainland, Donald, and Stewart, Chester B.: The Comparison of Percussion and Radiography in Locating the Heart and Superior Mediastinal Vessels, Am. Heart J. **15**: 515 (May) 1938.

SUMMARY

The determination of the fitness of the cardiovascular system for military service may be difficult because it is frequently necessary for the examiner to rely on signs alone and often these are of questionable significance. Tachycardia, systolic murmurs, slight elevation in blood pressure or a combination of these are the features that usually attract the attention of the examining physician. These are commonly of no importance, but they demand careful investigation. Thus tachycardia may lead to the disclosure of infection, particularly pulmonary tuberculosis. Moreover, in certain of the remaining, the response to exercise may be poor and further study may reveal the presence of an effort syndrome. Systolic murmurs should always be carefully evaluated. The history may give important, or even decisive, information. Finally, a decision regarding slight elevation of the blood pressure is not justified until the cardiac rate has receded to the normal level.

University Hospitals.

THE PROGNOSIS OF AURICULAR FIBRILLATION OF UNDETERMINED ORIGIN

FREDRICK A. WILLIUS, M.D.
AND
THOMAS J. DRY, M.B.
ROCHESTER, MINN.

From time to time, patients are encountered who display auricular fibrillation, either permanent or intermittent, as the sole evidence of disturbance of the heart. The frequent association of auricular fibrillation with obviously serious heart disease leads many physicians to consider this arrhythmia with gravity at all times and under all circumstances. Orgain and his co-workers [1] in 1936 called attention to the favorable prognosis associated with cases of uncomplicated or idiopathic auricular fibrillation. It is not our intention to cover the literature in this paper, for this was adequately done by Brill [2] in 1937.

Realizing the importance of acquiring further data on this subject in order to permit the segregation of the significant from the nonsignificant cases of auricular fibrillation, we have reinvestigated this subject with special reference to the clinical course of patients who exhibit auricular fibrillation in the absence of demonstrable evidence of organic heart disease and in the absence of any known etiologic factor.

From the Section of Cardiology, Mayo Clinic.

1. Orgain, E. S.; Wolff, Louis, and White, P. D.: Uncomplicated Auricular Fibrillation and Auricular Flutter: Frequent Occurrence and Good Prognosis in Patients Without Other Evidence of Cardiac Disease, Arch. Int. Med. **57**: 493-513 (March) 1936.

2. Brill, I. C.: Auricular Fibrillation: The Present Status with a Review of the Literature, Ann. Int. Med. **10**: 1487-1502 (April) 1937.

MATERIAL

Seventy patients observed on one or more occasions from 1920 to the time of this report (March 1941) comprised the basis for this study. The presence of auricular fibrillation, permanent or intermittently recurrent, was the primary basis of selection. From the resulting large group of records, those in which the following symptoms or signs were present were deleted: (1) cardiac enlargement or abnormalities of the cardiac silhouette as revealed by the roentgenogram, (2) cardiac murmurs, (3) hypertension, (4) hyperthyroidism, (5) electrocardiographic abnormalities such as significant changes in the T wave, disturbances in auriculoventricular or ventricular conduction and right axis deviation, and (6) observations obviously denoting heart disease. Patients more than 55 years of age were not included. Thus, patients included in this study showed auricular fibrillation as the sole evidence directing attention to the heart.

DISTRIBUTION ACCORDING TO AGE AND SEX

At the time at which the diagnosis of auricular fibrillation was made, the youngest patient was 26 years of age, the oldest was 55 and the average age for the group was 46.6 years. The distribution of the age of the patients by decades is shown in the table.

There was a decided preponderance of males over females, the ratio being 4:1. In the series reported by White and his co-workers an even larger preponderance of males occurred; the ratio was 7:1. The reasons for the discrepancy in incidence by sex are not clear.

FOLLOW-UP OF PATIENTS AFTER DIAGNOSIS

Of the 70 cases in the series reviewed we were able to obtain information regarding the present status of health in 59 either by means of recent examination or by correspondence. In what follows we shall refer only to these 59 patients.

Six patients were dead at the conclusion of this study; 5 of them died from causes entirely unrelated to the heart. The causes of death were, respectively, pneumonia, psoas abscess, carcinoma of the stomach, mushroom poisoning and noncardiac complications occurring after surgical repair of a large recurrent ventral hernia. Only 1 patient (1.7 per cent) died as the result of heart disease. This patient was a man for whom the diagnosis had been made when he was 55 years old and who died nine years later at the age of 64 years. This one death from heart disease among 59 traced patients is no more, and perhaps it is even less, than is to be expected in an average group of persons at the ages of the observed patients, considering the period of observation covered.

Fifty-three of the 59 patients who were traced were alive at the conclusion of this study. No significant differences were observed between the patients who had permanent auricular fibrillation (of which there were 30) and those who had intermittent auricular fibrillation (of which there were 29); therefore, all those alive at the end of the study will be considered in one category. Thirty-eight of the 53 patients (71 per cent) who were living reported their health as good and were able to engage in their accustomed occupations without difficulty. Eleven (21 per cent) of the 53 patients alive at the end of this study showed conditions entirely unrelated to the cardiovascular system, and 4 (8 per cent) were obliged to live under some restrictions: one because of the occurrence of slight edema when normal activities are pursued; another is known to be covered by disability insurance and in the case of the other 2 the cause of the disability is not definitely stated.

The full significance of these data is not appreciated until the time intervals of the study are considered. Twenty-three (43 per cent) of the 53 patients were alive from one to five years after their first examination at the Mayo Clinic, 13 (25 per cent) were alive from six to ten years after examination, 12 (23 per cent) were alive from eleven to fifteen years after examination, 3 (6 per cent) were alive from sixteen to twenty years after examination and 2 (4 per cent) were alive twenty years after examination.

The data were further analyzed with the assistance of the Division of Biometry and Medical Statistics of the Mayo Clinic. In the table are shown the ages of the patients at the time of diagnosis, and also the ages as of the time of last report. In this table it is seen that at the time of diagnosis the average age was 46.6 years and that, considering only the 59 patients who were traced in the follow-up study, the average age at the time of last report was 53.7 years. Therefore it is seen that the 59 patients in the traced group were observed for an average period of seven years. During this period there were six deaths, a mortality rate in this seven year period of about 10 per cent. For an average group of persons of age 47 years, according to United States Life Tables,[3] there is to be expected a mortality rate of about 9 per cent in the ensuing seven years, so that the number of deaths which had occurred in the group of patients suffering from auricular fibrillation was about what would be expected in a general group composed of persons of the same age observed for about the same length of time as our patients. It is certainly reasonable to suppose that, if these patients who had otherwise unexplained auricular fibrillation were subject to some form of latent cardiopathy at the time of the original examination, the adverse effects of this disease would become manifest in the period ensuing after discovery of the cardiac arrhythmia which was considered in this study.

Age at Diagnosis and at Last Report of Seventy Patients Who Had Auricular Fibrillation of Undetermined Origin

	Number of Patients at Stated Age			
	At Time of Diagnosis	At Time of Last Report		
Age, Years		Living	Dead	No Follow-Up
20-29	2	..	..	1
30-39	12	2	..	2
40-49	27	12	1	4
50-59	29	30	3	4
60+	..	9	2	..
Total	70	53	6	11
Mean	46.6	53.7		
Youngest	26			
Oldest	55			

IMPORTANCE OF TREATMENT IN THIS GROUP OF PATIENTS

It is of much interest to note that 35 of the 53 patients who were alive at the completion of the survey were not following any specific therapeutic regimen. Five of the remainder were taking quinidine in various amounts very irregularly, 6 were taking digitalis "off and on," and 7 gave but vague details of the treatment which they were receiving. The state of health of these

3. United States Life Tables, Department of Commerce, Bureau of the Census, 1930.

patients, then, obviously was not dependent on any rigid program of treatment. Experience has shown that, of all patients subject to auricular fibrillation, those under discussion are perhaps the most amenable to quinidine therapy, for in them a normal sinus rhythm can usually be established without much difficulty. Yet treatment with quinidine does not seem to be a life-saving measure for these patients and may be expected at the most to afford subjective comfort to a patient who is otherwise unduly conscious of his cardiac irregularity.

Paradoxically, the use of quinidine ("a potent drug, for good and for evil" as MacKenzie spoke of it) meets its greatest contraindications when it might be expected to do the most good; namely, among patients in whom an ectopic rhythm complicates an already seriously damaged heart.

CONCLUSIONS

The results of this study clearly indicate the benignity of auricular fibrillation occurring under the specified definition of this investigation and again emphasize the necessity for not venturing prognostic conclusions on the basis of the presence of this arrhythmia alone; rather, such conclusions should be based on the status of the heart as determined by the presence or absence of disease and on the functional capacity of the heart itself.

TREATMENT OF THE POSTENCEPHALITIC PARKINSONIAN SYNDROME

WITH DESICCATED WHITE WINE EXTRACT OF U. S. P. BELLADONNA ROOT

HOWARD D. FABING, M.D.
AND
MEYER A. ZELIGS, M.D.
CINCINNATI

Since the introduction of the "Bulgarian treatment" of postencephalitic parkinsonism by Raeff in 1926, numerous European and American investigators have confirmed the efficacy of this therapy in comparison with older methods of treatment.[1] Attempts have also been made to determine what factors in the treatment are responsible for the clinical improvement commonly noted in patients receiving the "Bulgarian treatment."

The basis of this treatment is the root of the belladonna plant. It has been held by some investigators [2] that the root of the belladonna plant indigenous to Bulgaria is therapeutically superior to roots grown elsewhere, but other investigators have denied this. Van Lehoczky [3] found no difference between Bulgarian and Hungarian varieties. Alcock and Carmichael,[4] Hill [5] and Neuwahl [6] found the English root as efficacious as the Bulgarian, and Vollmer [7] found that extracts of the Bulgarian and the U. S. P. roots could be interchanged in a given case without affecting the results. Panegrossi [8] used Italian roots, Baldauf,[9] German, Kauders and Oesterreicher,[10] Austrian and Coste and Devèze,[11] French, with results equal to those of the Bulgarian. Analysis of roots from various parts of the world tends to substantiate the clinical observation that the therapy is not dependent on the geographic source of the root. The alkaloidal content of the root does not differ qualitatively from region to region, although the gross appearance and total alkaloidal yield are variable.[12]

The method of extraction of the root, however, seems to be of great importance. Raeff, instead of employing dilute alcohol as an extractive and thereby obtaining an orthodox tincture, made a decoction of the macerated root using white wine as a menstruum. Pharmacologic studies by Neuwahl [6] and Fabing [13] have shown that there is a difference in the action of the hydroalcoholic extract (tincture) of belladonna root and the white wine extract. Fabing prepared decoctions of the root in identical fashion but used 12 per cent ethyl alcohol in one instance as an extractive and white wine in the other. Although the total alkaloidal yield was similar in the two products (0.02 per cent), the following differences in action were noted:

1. When 5 drops of the alcoholic extract was instilled in a cat's eye, intense mydriasis occurred. When the white wine extract was similarly employed, mydriasis did not result.

2. Subcutaneous injection of the two preparations in dogs led to different effects. Fifteen cc. of wine of commercial belladonna root was injected subcutaneously in a dog weighing 19 pounds (8.6 Kg.). The animal became drowsy and quiet and a bit depressed. The drug did not show any effect on respiration or on body temperature. There was some mydriasis about one hour after the injection. Fifteen cc. of a decoction prepared from the same commercial belladonna root and 12 per cent alcohol was injected subcutaneously in a dog weighing 20 pounds (9 Kg.). Shortly after injection the dog appeared uneasy and drowsy and showed signs of severe ataxia; there was enhanced panting, and the animal kept howling and barking; there was intense mydriasis. The animal remained in its excited and apparently delirious state for more than twelve hours and showed a recovery to normal in the next twenty-four hours.

Thus pharmacologic differences in the mydriatic effect in the cat's eye and the toxic effects on sub-

From the Department of Internal Medicine, Neurologic Division, University of Cincinnati College of Medicine.

1. Ferrannini, Luigi: La cura bulgara di alcuni postumi di encefalite letargica, Policlinico (sez. prat.) **42:** 967-974 (May 20) 1935. von Witzleben, H. D.: Die Behandlung der chronischen Encephalitis epidemica mit der bulgarischen Kur, Psychiat.-Neurol. Wchnschr. **39:** 403-404 (Sept. 4) 1937; **40:** 73 (Feb. 12) 1938. von Witzleben, H. D., and Werner, A.: Behandlung der chronischen Encephalitis epidemica (Parkinsonismus): Bulgarische Kur mit "Homburg 680," Deutsche med. Wchnschr. **64:** 1174-1178 (Aug. 12) 1938. Vollmer, Hermann: The Bulgarian Treatment of Postencephalitic Parkinsonism, J. Mt. Sinai Hosp. **6:** 93-99 (July-Aug.) 1939. van Lehoczky,[3] Alcock and Carmichael,[4] Fabing.[13]

2. Neuwahl, F. J.: Treatment of Parkinsonism with Bulgarian Belladonna, Lancet **1:** 693-695 (March 25) 1939. von Witzleben, H. D.: Die Behandlung der chronischen Encephalitis epidemica (Parkinsonismus) mit der "bulgarischen Kur," Klin. Wchnschr. **17:** 329 (March 5); 369 (March 12) 1938.

3. van Lehoczky, T.: Behandlung bei Parkinsonismus postencephaliticus. (Bericht über 64 Fälle), Deutsche Ztschr. f. Nervenh. **146:** 263-271, 1938.

4. Alcock, N. S., and Carmichael, E. A.: Investigation into Treatment of Parkinsonism with Bulgarian Belladonna, Quart. J. Med. **7:** 564-574 (Oct.) 1938.

5. Hill, D.: Bulgarian Treatment: Comparison with English Belladonna, Lancet **2:** 1048-1050 (Nov. 5) 1938.

6. Neuwahl, F. J., and Fenwick, C. C.: Bulgarian Treatment of Parkinsonism, Lancet **2:** 619-621 (Sept. 11) 1937.

7. Vollmer, Hermann: "Bulgarian Treatment" of Parkinson's Disease, Arch. Neurol. & Psychiat. **43:** 1057-1080 (June) 1940.

8. Panegrossi, G.: Ueber die neue Heilmethode der chronischen epidemischen Enzephalitis mit Parkinson-Erscheinungen, Deutsche med. Wchnschr. **64:** 669-670 (May 6) 1938.

9. Baldauf, F.: Zur Frage der Behandlung postenzephalitischer Zustände (Parkinsonismus) mit deutscher Belladonnawurzel. (Vorläufiger Bericht über ein neueres biologisches Verfahren), München. med. Wchnschr. **85:** 54-57 (Jan. 14) 1938.

10. Kauders, O., and Oesterreicher, W.: Ueber die Wirksamkeit der sogenannten bulgarischen Kur beim postenzephalitischen Parkinsonismus im Vergleiche zur Behandlung mit hohen Atropindosen, Wien. klin. Wchnschr. **49:** 1548-1552 (Dec. 18) 1936.

11. Coste, M., and Devèze, M.: A propos du traitement des syndromes postencéphalitiques par la "cure bulgare," Bull. et mém. Soc. méd. d. hôp. de Paris **52:** 814-815 (May 25) 1936.

12. Bailey, A. E.: Bulgarian Belladonna Root, Pharm. J. **140:** 567-568 (May) 1938. Taylor, S. A., and Hobart, F. G.: Bulgarian Belladonna, ibid. **141:** 49-51 (July) 1938. Bulgarian Belladonna, editorial, Pharm. J. **141:** 625-626 (Dec. 17) 1938.

13. Fabing, H. D.: The Bulgarian Belladonna Treatment of Postencephalitic Parkinsonian Syndrome, Ohio State M. J. **35:** 1195-1199 (Nov.) 1939.

cutaneous injection in dogs seem to point to a qualitative rather than to a quantitative difference of alkaloidal yield when the same belladonna root is extracted with 12 per cent alcohol and with white wine.

A synthetic product [14] has been developed in this country to approximate the alkaloids found in white wine extract of belladonna root, and clinical reports concerning its use have been favorable.[15] Neal and Dillenberg,[16] however, found that this preparation yielded clinical improvement which was similar in nature but not as well defined as that obtained with a "natural" white wine decoction of Bulgarian belladonna root.

METHODS AND MATERIALS

The drug employed in the treatment of the patients with parkinsonism was a white wine extract of U. S. P. belladonna root. One pound (454 Gm.) of ground U. S. P. belladonna root was mixed with five pints (2,365 cc.) of dry white wine and allowed to stand overnight at room temperature. After the extraction, the filtrate was concentrated under reduced pressure until the product was reduced from its liquid state to a pilular consistency. The product was then chemically assayed and adjusted so that each 100 Gm. contained 0.75 Gm. of the total alkaloids of belladonna root. The extract was made into a granulation by mixing with an inert material and drying at a temperature not exceeding 140 F. (60 C.). The granulated mass was assayed chemically, and further inert material was added so that each tablet made from it would contain 0.4 mg. of the total alkaloids. The granulated mass was compressed into tablets, which were given a light sugar coating.[17]

CLINICAL MATERIAL

The patients included in this study were selected from office practice and from the wards of the Hamilton County Chronic Disease Hospital. The period of observation extended throughout the year 1940 and in many cases through 1939. There was a total of 64 patients, of whom 13 were in institutions and the remainder were studied in private practice. These patients exhibited all grades of severity of the parkinsonian syndrome. The duration of symptoms varied from two to fifteen years.

The method of administering the drug was to begin with small doses, usually 1 tablet at bedtime. The dose was then raised slowly to the level of each individual patient's tolerance. The level of tolerance varied widely, from 3 to 50 tablets a day. The average optimal dose was found to be 7 to 10 tablets daily. A too rapid rise in the dose was avoided, and a month or more was usually employed in determining the final optimal dose. Some patients were found to do best when taking but two doses daily (at bedtime and twelve hours later in the morning), whereas others best tolerated three doses daily at intervals of eight hours, and still others seemed to profit most by dividing their total dose into four or five portions given throughout the day. The optimal dose was defined as that dose which brought the greatest clinical relief with the least toxic side reactions. Since the margin between the most effective therapeutic dose and the toxic dose was always a small one, it was necessary to observe these patients carefully during the trial period while the optimal dose was being determined. The number of tablets necessary to bring about relief of symptoms usually varied in accordance with the severity of the parkinsonism; larger doses had to be employed in the cases of more severe parkinsonism.

TOXIC EFFECTS

Toxic effects were common and were essentially the same as those found in poisoning with any drug of the atropine series. The earliest toxic effects were moderate mydriasis, difficulty in accommodation and dryness of the upper respiratory passages. Increase in the dose caused increase in these symptoms, plus flushing, redness and hotness of the skin, slight tachycardia and suppression of sweating. Cerebral symptoms beginning with dizziness and some clouding of the sensorium and then even progressing to hallucinosis and confusion may occur if the dose is increased indiscriminately. Therefore any symptoms beyond mild mydriasis and dryness of the mouth should be met by dropping the dose slightly and maintaining it at the lowered level until a new level of tolerance has developed. The root of the sweet iris, or calamus root, has been advocated to counteract the oral dryness which almost universally accompanies use of the drug, but most patients have found calamus root so bitter that they have preferred the dryness to such a taste. Hard lemon-flavored candy drops have proved useful in combating dryness of the mouth. Instillation of 1 to 2 drops of 1 per cent pilocarpine hydrochloride solution into each eye has proved effective in relieving mydriasis and disturbances of ocular accommodation, especially in those patients who read a great deal.

Degree of Improvement in Severe and Moderate Parkinsonism

	Improvement										
	Maximal ++++		Moderate +++		Slight ++		Minimal +		None 0		
Previous Condition	No.	%	No.	%	No.	%	No.	%	No.	%	Total
Moderately severe	18	54.5	15	45.5	0	0.0	0	0.0	0	0.0	33
Very severe	8	25.8	10	32.2	8	25.8	3	9.7	2	6.5	31
Total	26	40.6	25	39.0	8	12.5	3	4.7	2	3.2	64

The management of patients with parkinsonism is far easier in an institution than it is in office practice. In the latter instance the patients are seen at infrequent intervals and occasionally a mild toxic state develops which is upsetting to the patient and his household. A mild sedative effect is common with this medication, and these patients tend to sleep at slightly longer intervals at night, and many of them like to take a nap in the afternoon, although they had not been accustomed to do so formerly.

No dietary restrictions seemed necessary, but all patients reported that alcoholic drinks cannot be tolerated with the drug. Patients frequently reported acute gastric upsets after a single whisky highball or after a single cocktail. It is generally true, although not universally so, that patients who can tolerate large quantities of belladonna are those who have tolerated other drugs of the atropine series in large quantities. Patients who begin using this drug come to depend on it and are faithful in taking their medication. It has been

14. "Rabellon," a product of Sharp & Dohme, Philadelphia.
15. Gayle, R. F., Jr.: Treatment of Parkinsonism with a Preparation of Belladonna Root, Virginia M. Monthly **66**: 707-710 (Dec.) 1939. Vollmer.[7]
16. Neal, Josephine B., and Dillenberg, S. M.: A Comparison of Bellabulgara and Other Forms of Medication in the Treatment of Chronic Encephalitis, New York State J. Med. **40**: 1300-1302 (Sept. 1) 1940.
17. "Vinobel," a product of the William S. Merrell Company, Cincinnati.

reported that after some months the drug can be discontinued and the gains made will be maintained. This, most emphatically, has not been our experience.

CLINICAL RESULTS

The results were impressive and approximated those found by other workers with belladonna therapy. The symptoms most improved were those of rigidity, salivation and postural disturbances, such as posteropulsions and anteropulsions. Relaxation of muscular rigidity and a general subjective feeling of well-being usually heralded the beginning of improvement. The rigidity was more beneficially affected than the tremor, the latter being less frequently improved unless it was accompanied by some degree of muscular stiffness. Oculogyric crises were relieved entirely in 3 cases and improved in 2 cases.

"Cure" is a term which implies a healing of the underlying lesion, and this is not to be expected. The evaluation of improvement in a therapeutic investigation in which no quantitative laboratory methods are available is more than difficult, for it is totally dependent on the arbitrary appraisal of the observer. In order to describe more accurately the degree of improvement noted in these patients, we have set up the following criteria for various levels of improvement:

1. "Minimal" (+) = subjective improvement and some lessening of nursing care.

2. "Slight" (++) = definite improvement in ability to get around and feed self; nursing care no longer necessary.

3. "Moderate" (+++) = the foregoing plus objective improvement in rigidity and/or tremor, postural disturbances, gait, speech and facies plus partial economic and social rehabilitation.

4. "Maximal" (++++) = the foregoing plus a complete or almost complete clinical abatement of parkinsonian symptoms plus complete or almost complete economic and social rehabilitation.

The patients were divided on the basis of the degree of neurologic involvement into groups with moderately severe and very severe parkinsonism. The results, grouped in accordance with the foregoing standards, were tabulated as is shown in the accompanying table.

COMPARISON OF THERAPEUTIC EFFECT OF BULGARIAN AND U. S. P. EXTRACTS

Of the total 64 patients, 46 were treated with a similarly prepared liquid white wine decoction of belladonna root gathered in Bulgaria and obtained through Paul Hopfe of Hamburg, Germany, who guaranteed its source, before they were treated with the desiccated U. S. P. tablet. In no case were we able to determine any difference in therapeutic effect, and the two methods of therapy have been interchanged without alteration of results. Since the liquid preparation was found to be chemically unstable after a short period of time, it was replaced by the desiccated tablet.

SELECTION OF PATIENTS

It is impossible to state which patients will respond best to the treatment, but it is generally true that those under 40 years of age, those whose symptoms have been present but a few years, those with considerable rigidity and little tremor and those who can tolerate large quantities of the drug will do best. Those with involvement on one side only usually do well also. However, the variability in response is so great that one is justified in attempting therapy with the drug even when these ideal conditions are not present.

The drug is effective in the cases of postencephalitic parkinsonism as well as in a large group of cases in which no clear history of acute encephalitis is available but in which involvement is almost certainly of postencephalitic origin. However, patients with the so-called degenerative type of the disease who have associated cerebral arteriosclerosis and generalized vascular disease and who are usually above 60 years of age are unable to tolerate large enough quantities of the drug to receive benefit at all comparable to that in the postencephalitic group. Some few of these patients, however, are helped, and the drug deserves to be tried in cases of degenerative parkinsonism. It must be borne in mind that toxic symptoms occur quickly in these persons with arteriosclerosis, and one must be prepared to abandon the drug if untoward effects occur with small doses.

SUMMARY

Since the introduction of the "Bulgarian belladonna treatment" of postencephalitic parkinsonism by Raeff in 1926, many investigators have confirmed the efficacy of this therapy. Study of the factors in the treatment which are responsible for the clinical improvement so often seen in cases of this type of parkinsonism reveals that there is no peculiar efficacy in the belladonna root grown in Bulgaria but that the apparently better results are due to the manner of extracting the alkaloids of the root. White wine extracts seem to yield products qualitatively different from those obtained by hydroalcoholic extraction (tinctures). This statement is confirmed by pharmacologic study. A preparation of a white wine extract of U. S. P. belladonna root which was then desiccated and pressed into tablets containing a total alkaloidal content of 0.4 mg. each was used.

Of a total of 33 patients with moderately severe parkinsonism 54.5 per cent showed a maximal improvement and 45.5 per cent showed a moderate improvement. Of a total of 31 with very severe parkinsonism 25.8 per cent showed a maximal improvement, 32.2 per cent showed a moderate improvement, 25.8 per cent showed a slight improvement, 9.7 per cent showed a minimal improvement and 6.5 per cent showed no improvement.

The desiccated tablet made from U. S. P. belladonna root was found to be as efficacious therapeutically as was a white wine decoction of the Bulgarian belladonna root. The dose was found to vary widely. Toxic reactions were frequent, and the dose had to be varied to prevent the occurrence of these reactions. In cases of arteriosclerotic parkinsonism, the drug was found to be too toxic for general use.

It is felt that preparations of the type described are superior to drugs of the belladonna series heretofore employed in the treatment of postencephalitic parkinsonism.

Oxygen.—Joseph Priestley (1733-1804), a nonconformist English clergyman, is usually credited with the first isolation of oxygen. His results were published in 1774. The gas was obtained by heating mercuric oxide and isolated by collection over mercury in a pneumatic trough. He recognized the fact that substances burned more brilliantly in this gas than in air and that it supported life. However, he failed completely to realize that the true explanation of the nature of combustion was in his hands. Even after his meeting with Lavoisier he remained unconvinced. He predicted the use of oxygen in medicine.—Timm, John A., in Development of the Sciences, New Haven, Conn., Yale University Press, 1941.

THE TREATMENT OF PARKINSONISM

RESULTS OBTAINED WITH WINE OF BULGARIAN BELLADONNA AND THE ALKALOIDS OF THE U. S. P. BELLADONNA

J. C. PRICE, M.D.
AND
H. HOUSTON MERRITT, M.D.
BOSTON

Ivan Raeff, of Chipka, Bulgaria, a biologist, in 1926 made a decoction of the root of the Bulgarian belladonna plant with wine and instituted an elaborate procedure for the treatment of parkinsonism. Most astonishing results were claimed. Since Raeff's study there have been many reports in the literature on the use of Bulgarian belladonna.[1] Elaborate side measures which were part of the therapeutic program of some investigators included change in environment, special nursing care, physical therapy, systematic exercises and occupational therapy. In addition, the psychologic effect of the sympathy and attention given the patients must have been considerable.

It has been stated that in the treatment of patients with parkinsonism Bulgarian belladonna has a value superior to that of the belladonna grown in other countries. This claim has not been supported by the results of investigators using the English, Italian or German root. It was the purpose of our study to investigate the therapeutic effect of the wine decoction of Bulgarian belladonna on the symptoms of parkinsonism and to determine whether there was any specific action of the white wine decoction of the Bulgarian belladonna which could not be obtained with the alkaloids of the U. S. P. belladonna or with the Bulgarian belladonna administered in forms other than the white wine decoction. With these aims in view we treated 71 patients with the following preparations: (1) white wine extract of Bulgarian belladonna root;[2] (2) desiccated white wine extract of U. S. P. belladonna root,[3] and (3) tablets containing a combination of the alkaloids of the belladonna root (hyoscyamine, atropine and scopolamine).[4]

In addition, the Bulgarian belladonna was given in a hydroalcoholic extract [2] and the U. S. P. belladonna in both white wine extract [2] and hydroalcoholic extract.[2]

The patients used in this study all were suffering from parkinsonism, and no attempt was made to select them according to the severity of the symptoms or the etiologic factors in their disease. The treatment was given in the wards or the outpatient department of the Neurological Unit of the Boston City Hospital or in the wards of the Long Island Hospital (the city hospital for patients with chronic diseases). An attempt was made to keep the environment during the study the same as that prior to the administration of treatment. Physical therapy was not given, and encouragement or other forms of psychotherapy were avoided as far as possible.

METHOD OF ADMINISTRATION

The treatment was started with a dose of 0.1 to 0.2 mg. given one to four times a day. This dose was increased by increments of 0.1 to 0.2 mg. at intervals of one to three days until the appearance of untoward side reactions made further increases inadvisable. The optimum dose was then determined by gradually decreasing the total daily amount until a dose was obtained which gave the maximum relief from the symptoms of the parkinsonism with a minimum or tolerable amount of side symptoms.

RESULTS

The degree of relief of symptoms was graded from "none" to "maximal," as follows: "minimal," subjective improvement and some lessening of nursing care; "slight," definite improvement in ability to get around and feed self, and much less need for nursing care; "moderate," the foregoing, with the addition of objective improvement in rigidity, tremor, speech, facies, posture and partial economic and social rehabilitation; "maximal," complete or almost complete clinical abatement of symptoms with complete or almost complete economic and social rehabilitation.

The results obtained with the three preparations which formed the major part of this study are shown in table 1. These results indicate that there was little difference in the therapeutic effects of the three preparations. The figures shown in this table could be interpreted as indicating that the best results were obtained with the desiccated white wine extract of U. S. P. belladonna, but the relatively small number of cases and other factors must be taken into consideration; for example, many of the patients treated with the compound of belladonna alkaloids were chronically bedridden. It was found, furthermore, that the majority of the patients could be changed from one preparation to

From the Neurological Unit, Boston City Hospital, and the Department of Neurology, Harvard Medical School.

The wine and hydroalcoholic extracts of both the Bulgarian and the U. S. P. belladonna root and the desiccated white wine extract of U. S. P. belladonna were furnished by the William S. Merrell Company and the compound of the belladonna alkaloids by Sharp & Dohme.

1. These reports include:

Bailey, A. E.: Bulgarian Belladonna Root, Pharm. J. **140:** 77 and 567, 1938.

Fabing, H. D.: Bulgarian Belladonna Treatment of Postencephalitic Parkinsonian Syndrome, Ohio State M. J. **35:** 1195, 1939.

Ferrannini, L.: La cura bulgara di alcuni postumi di encefalite letargica, Policlinico (sez. prat.) **42:** 967 and 1544, 1935.

Forster, F. M.: Treatment of Parkinsonian Syndrome, Pennsylvania M. J. **43:** 67, 1939.

Gayle, R. F., Jr.: Treatment of Parkinsonism with a Preparation of Belladonna Root: Preliminary Report, Virginia M. Monthly **66:** 707, 1939.

Henricksen, R. H.: Bulgarian Belladonna, Pharm. J. **140:** 240, 1938; **141:** 522, 1938; Bulgarian Treatment of Postencephalitic Parkinsonism, Lancet **2:** 1141, 1938.

Hill, D.: Bulgarian Treatment of Postencephalitic Parkinsonism: Comparison with English Belladonna, ibid. **2:** 1048, 1938.

Lewenstein, H.: Die Behandlung der Folgezustände der Encephalitis epidemica mit hohen Atropindosen, Deutsche med. Wchnschr. **57:** 1014, 1931.

di Mattei, P.: Sulla cosi detta "cura bulgara" dei postumi di encefalite epidemica, Policlinico (sez. prat.) **42:** 501, 1935.

Neal, J. B.: Bulgarian Belladonna Treatment of Chronic Encephalitis: Preliminary Report, New York State J. Med. **39:** 1875, 1939.

Neuwahl, F. J.: Treatment of Postencephalitic Parkinsonism with Bulgarian Belladonna, Lancet **1:** 693, 1939.

Neuwahl, F. J., and Fenwick, C. C.: Bulgarian Treatment of Postencephalitic Parkinsonism, ibid. **2:** 619, 1937.

Panegrossi, G.: Ueber die neue Heilmethode der chronischen epidemischen Enzephalitis mit Parkinson-Erscheinungen, Deutsche med. Wchnschr. **64:** 669, 1938.

Roemer, C.: Zur Atropinhandlung der enzephalitischen Folgezustände, München. med. Wchnschr. **77:** 2156, 1930; Zur Therapie der enzephalitischen Folgezustände (Atropin in hohen Dosen, Atropin-Pilokarpin, Eserina), Med. Klin. **28:** 224, 1932; Die Atropinbehandlung des Parkinsonismus, Med. Welt **6:** 1127, 1932.

Taylor, S. A., and Hobart, F. G.: Bulgarian Belladonna, Pharm. J. **141:** 49, 1938.

Tocco, L.: Osservazioni e richerche sulla cura bulgara del parkinsoniano postencefalico, Rassegna di terap. e pat. clin. **8:** 154, 1936.

Vollmer, Hermann: Bulgarian Treatment of Postencephalitic Parkinsonism, J. Mt. Sinai Hosp. **6:** 93, 1939; Bulgarian Treatment of Parkinson's Disease: Pharmacologic Aspects and Clinical Effects of Alkaloids of Belladonna Root, Arch. Neurol. & Psychiat. **43:** 1057 (June) 1940.

von Witzleben, H. D.: Die Behandlung der chronischen Encephalitis epidemica (Parkinsonismus) mit der "bulgarischen Kur," Klin. Wchnschr. **17:** 329 and 369, 1938.

2. A white wine extract or hydroalcoholic extract of both the Bulgarian and the U. S. P. belladonna root prepared by the Wm. S. Merrell Co. after the method of Neuwahl and Fenwick to contain not more than 0.0173 Gm. or less than 0.0132 Gm. of total alkaloid per hundred cubic centimeters.

3. White wine extract of U. S. P. belladonna prepared by Wm. S. Merrell Co. in the same manner as described, desiccated and pressed into tablets containing 0.4 mg. or 0.8 mg. of total alkaloid, marketed under the trade name of "Vinobel."

4. Alkaloids of U. S. P. belladonna combined in tablet form, each tablet containing hyoscyamine 0.4507 mg., atropine sulfate 0.0372 mg. and scopolamine hydrobromide 0.0119 mg., marketed under the trade name "Rabellon" by Sharp & Dohme.

another without appreciable difference in the therapeutic results. Nine of the 71 were treated at different times with each of the three preparations, and, although there were some individual variations, the therapeutic results were practically the same with all three preparations (table 2).

The hydroalcoholic extract of Bulgarian belladonna and wine and hydroalcoholic extract of U. S. P. belladonna were administered to 8 patients both as initial and as substitutive therapy for wine of Bulgarian belladonna. Initial and substitutive therapy with these two preparations gave therapeutic and toxic side effects identical with those obtained with the white wine extract of Bulgarian belladonna.

TABLE 1.—*Results of Treatment of Parkinsonism*

Medication	Severity of Symptoms	Degree of Improvement					
		None	Minimal	Slight	Moderate	Maximal	Total
Wine of Bulgarian belladonna	Mild	3	7	1	2	4	17
	Severe	9	13	13	8	0	43
Desiccated white wine extract of U. S. P. belladonna	Mild	0	1	0	1	4	6
	Severe	3	12	5	1	2	23
Compound of belladonna alkaloids	Mild	0	2	2	1	2	7
	Severe	6	6	2	4	1	19
Total	Mild	3	10	3	4	10	30
	Severe	18	31	20	13	3	85

Although it is known that patients suffering from parkinsonism are apt to experience subjective improvement with any new treatment, an attempt was made to compare the results obtained in this study with those previously obtained in the same patients with their former modes of therapy (usually stramonium pills or scopolamine hydrobromide tablets). The results as given by 45 patients were (1) no better 15, (2) worse 5 and (3) better 25. Thirty-four of these 45 patients had been under our care for several to many years and had been given various therapeutic agents. Five obtained a greater degree of improvement with the new treatment, 28 obtained equivalent results and 1 was made worse. Former therapy had been definitely effective in 8 and of no benefit in 2 of the 14 patients who were moderately or maximally improved with the wine of Bulgarian belladonna (table 1). The results in 4 could not be compared. Former therapy had been ineffective in 10 of the 12 patients who failed to respond to wine. In 1 the treatments could not be compared and 1 had been moderately relieved by atropine.

In the analysis of our results the following factors seemed to be of importance in regard to the effects of treatments:

1. Cause of the symptoms: Benefit from the treatment was obtained by 89 per cent of the patients with a history of encephalitis preceding the onset of symptoms as compared to 24 per cent of those whose symptoms presumably were due to arteriosclerosis.

TABLE 2.—*Results of Treatment with All Three Preparations in Nine Cases*

Patient	Age	Duration of Symptoms	Cause of Symptoms	Severity of Symptoms	Degree of Improvement with		
					Wine of Bulgarian Belladonna	Desiccated White Wine Extract of U. S. P. Belladonna	Compound of Belladonna Alkaloids
1. L. B.	35	19	Encephalitis	Severe	Slight	Slight	Slight
2. H. B.	51	10	Encephalitis	Severe	Slight	Minimal	Slight
3. G. C.	58	2	Arteriosclerosis	Moderate	Minimal	Minimal	Slight
4. R. E.	56	9	Unknown	Moderate	Maximal	Maximal	Maximal
5. P. G.	50	10	Encephalitis	Severe	None	Minimal	Minimal
6. P. K.	56	3	Arteriosclerosis	Moderate	Maximal	Moderate	Moderate
7. K. M.	56	2	Arteriosclerosis	Moderate	Maximal	Maximal	Maximal
8. M. S.	41	19	Encephalitis	Severe	Moderate	Maximal	Maximal
9. M. Y.	66	1	Arteriosclerosis	Severe	None	None	None

The use of dry white wine, carrier of the decoction preparations, as initial therapy failed to alter the symptoms of parkinsonism of 4 patients, and substitution therapy with the white wine caused a regression to the pretreatment status of 2 patients who had responded favorably to therapy with wine of Bulgarian belladonna, and there was no change in the condition of 2 patients who had failed to respond to the therapy with white wine of Bulgarian belladonna.

Since the results of these various compounds on the total picture and the individual symptoms of the disease were almost identical, there is no need for a separate discussion in summarizing our results. Doses when given are in milligrams of total alkaloid.

The results obtained in the 71 cases can be summarized as follows: no improvement 20 per cent, minimal improvement 31 per cent, slight improvement 25 per cent, moderate improvement 17 per cent and maximal improvement 7 per cent.

The treatment was discontinued in 17 cases, and there was a regression to the pretreatment status in 16 of the 17 within ten days, sometimes developing as early as twenty-four hours but usually in two to five days. One patient whose tremor had been relieved by treatment continued to be free of the tremor after the treatment was withdrawn. He had had several spontaneous remissions of the tremor in the past.

2. Age at onset of symptoms: Eighty-five per cent of the patients with onset of symptoms before the age of 40 were improved as contrasted to 70 per cent in the group with onset after the age of 40.

3. Duration of symptoms: The average duration of symptoms in the group of patients who were improved by the treatment was seven years as compared to twelve years in the group whose symptoms did not respond to treatment.

4. Character of symptoms: As a rule the symptoms of excessive salivation and a subjective sense of muscular rigidity almost always responded to treatment. Objective muscular rigidity, "cogwheel" phenomena, with associated poverty of motion, propulsions or retropulsions, flexor habitus, oculogyric crises and tremor responded well to the treatment in some instances and in others were little affected. Abnormal speech, masklike facies, tics and loss of associated movements were the symptoms which were most difficult to influence.

When the major part of the clinical syndrome was rigidity the results were more favorable. Improvement was obtained in 85 per cent of the group with this symptom as compared to 65 per cent of the group in which tremor was the principal clinical manifestation.

TOXICITY

The amount of the wine of Bulgarian belladonna which could be taken by the patients varied widely, namely between 0.4 and 12 mg. a day. The average maximum dose in the 60 cases was 3.3 mg. The level at which untoward side symptoms developed varied between 0.2 and 2.7 mg., with an average of 1 mg. Larger doses were tolerated by the patients whose symptoms developed as a sequel to encephalitis than by those whose symptoms were due to arteriosclerosis.

One of the experimental and clinical advantages which has been claimed for wine of Bulgarian belladonna is a low toxicity. The relative toxicity of other belladonna preparations used in this study was determined in 38 cases. The level at which toxic symptoms appeared and the maximum tolerated doses were exactly similar for all preparations in all patients, with 1 exception. This patient was able to tolerate only small doses of the wine extract of Bulgarian belladonna but could take large doses of tincture of U. S. P. belladonna.

NATURE OF TOXIC SYMPTOMS

Dryness of the mouth was the most frequent complaint, occurring in 88 per cent of the cases. Blurring of vision was second, with an incidence of 68 per cent. With increase in the dose, urinary retention and impairment of pupillary response to light and accommodation develop. Less frequent toxic manifestations were diplopia, conjunctivitis, photophobia, dysarthria, dizziness, headache, mental confusion, glossitis, dysphagia, gastralgia, diarrhea and constipation. A sense of internal heat and flushing of the skin was infrequent. Blood pressure may be affected by large doses with a decrease in the systolic pressure and an increase in the diastolic pressure. Acute infectious diseases lower the tolerance of the patient, as does excessively hot weather. One patient suffered a "heat stroke" after direct exposure to the sun for one hour in midsummer. Arteriosclerotic heart disease, angina pectoris, severe general arteriosclerosis, mental disturbances, chronic nephritis, glaucoma, malnutrition, general debility and dehydration contraindicate the use of the belladonna drugs.

Three of the original 71 patients have died. A young man with severe symptoms committed suicide by jumping from a seventh story window. A middle-aged woman with severe symptoms was found dead in bed one morning. She had had no therapy for a period of four to five days. A middle-aged Chinese with severe symptoms succumbed to repeated gastric hemorrhages while taking wine of Bulgarian belladonna in the dose of 1.6 mg. daily. Postmortem examination revealed no anatomic or pathologic explanation for the hemorrhages.

Treatment of the toxic symptoms does not yield good results. Blurring of vision may be helped by the use of lenses correcting the disturbance in accommodation. Dryness of the mouth is combated by constantly keeping rock candy, lemon drops, a small pebble or chewing gum in the mouth. The drugs available for treatment of these symptoms are only moderately efficient and have undesirable effects in themselves. Physostigmine as an ophthalmic ointment or as an aqueous solution of 0.25 to 0.5 per cent may be used for blurring of vision, but local irritation prevents prolonged use. Pilocarpine hydrochloride (1 per cent solution) may also be used. Twitching of the intrinsic muscles of the eye may become annoying. Pilocarpine hydrochloride, in the dose of $\frac{1}{12}$ to $\frac{1}{6}$ grain (0.005 to 0.01 Gm.) given orally or hypodermically, has been tried, but the occurrence of severe diarrhea, profound general weakness, profuse sweating and headache makes its use undesirable. Care must be taken not to use pilocarpine in the presence of "shock" as seen in "heat stroke."

SUMMARY

1. Wine and hydroalcoholic extracts of Bulgarian and U. S. P. belladonna root, desiccated white wine extract of U. S. P. belladonna and a compound of the belladonna alkaloids were used in treatment of 71 unselected patients with parkinsonism.

2. The results obtained with the three preparations were practically identical.

3. Among 30 patients with symptoms of a mild degree of severity the results were as follows: not improved, 10 per cent; moderately improved, 43 per cent, and greatly improved, 47 per cent. The results obtained on 85 patients with symptoms of severe degree were 22 per cent not improved, 59 per cent moderately improved and 19 per cent greatly improved.

4. The age of onset of 40 years or less, the duration of the syndrome of ten years or less or the history of encephalitis preceding the onset of symptoms enhance the probability of a favorable response.

5. Dryness of the mouth, blurring of vision, impairment of pupillary response to light, urinary retention and impairment of pupillary response in accommodation are the most frequent toxic symptoms. An increase in the dose beyond the level producing these symptoms may cause mental confusion, dysarthria, dysphagia, headache, diplopia, diarrhea or constipation.

6. Contraindications to the use of these drugs are advanced arteriosclerotic disease of the heart or the peripheral vessels, disturbances of the mental condition, chronic nephritis, glaucoma, malnutrition and dehydration.

7. The alkaloids derived from the root of the belladonna plant irrespective of its geographic origin are valuable in the treatment of parkinsonism.

PLASTIC FLAP IN SURGERY OF THE TONSILS

ROBERT H. FOWLER, M.D.
NEW YORK

Today the surgeon in almost every instance can assure the patient that the tonsils will be successfully removed—the tonsil, the whole tonsil and nothing but the tonsil—without injury to the other structures of the throat; yet four decades ago such assurance was the exception rather than the rule. Up to the turn of the century tonsillotomy (partial removal) was practiced, and the use of that operation continued through the first decade; in the second decade of this century too much was removed; automatic instruments came into vogue which stripped away from the throat a thin sheet of muscle; in the third and fourth decades standards of removal of the tonsils have been improved until a "muscle free dissection" is the common aim of all.

MUSCLE FREE DISSECTION

Anatomic research has shown that the tonsil is not a spherical cyst with a thick capsule, as has been believed, but is instead a complicated double pocket composed of two lobes marked off from each other by an hourglass constriction. The so-called capsule is an artefact, the first layer stripped from the fascia between the tonsillar tissue and muscles. The fascia may be regarded as a fine network of fibrous bands. It is the undifferentiated areolar tissue between the well defined

lymphoid tissue of the tonsil and the equally well defined muscle tissue lateral to it. Instead of having a ready-made capsule, the lymph-lined double pocket which is the tonsil is closely bound to the muscles of the pillars and firmly attached to them by strong fibers of the

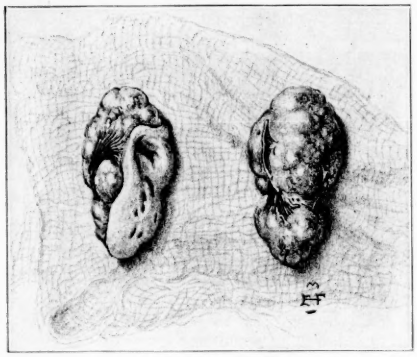


Fig. 1.—Corpus delicti. Quilted appearance of surface of tonsil after removal by meticulous "muscle free dissection."

pharyngeal fascia. Over the upper lobe or pocket the layers of this pharyngeal fascia are easily split apart in the process of dissection over the submerged surface of the upper lobe. For this reason the first half of the operation runs off smoothly, but the dissection of the lower half of the tonsil, the hourglass constriction and the submerged surface of the lower lobe, is more difficult; it requires greater force and more accurate study for a definite reason: In this area the muscles are firmly anchored to the tonsil, their fascial fibers terminating between the lobules in the connective tissue trabeculae.

In figure 1 is shown a pair of tonsils which have been gently removed and freshly laid on a piece of gauze. Just below the middle is seen the hourglass constriction which marks off the large upper lobe or pocket from the smaller lower lobe or pocket, and the attachments of the muscle are shown cut short against the surface of the tonsil. The appearance of the surface of both upper and lower lobes is that of being quilted because of the pull of the trabeculae when only a single intimate layer of the submucosa remains to cover the lymphoid tissue of the tonsil. This quilted appearance of the fascia on the surface of the tonsil after removal and the short stumps of the muscle at the hourglass constriction are evidences of what has been a successful "muscle free dissection."

PLASTIC FLAPS IN SURGERY OF THE TONSILS

For fifty years we have seen the tonsillar fossa neglected after all traditional operations and the whole raw surface of the wound after enucleation left exposed to every accident of trauma from food in the throat and to its full quota of infection. There is something fundamentally wrong here.

It takes but a minute to cut a flap at the time of the first incision of the mucous membrane; it takes less than a minute when the tonsil has been removed to anchor this flap with a catgut slipknot to the fascia at the center of the wound in such a way as to help nature in the healing. But the technic must be accurate; it takes a deft hand, and the new design must be based on anatomic lines which nature follows in the repair. There must be no sewing together of the two pillars. Each muscular pillar can be promptly protected with mucosa at the time of operation.

REPAIR OF THE TONSILLAR FOSSA

The new plan of repair designed to minimize convalescence by partially covering the exposed portion of the pillars with a protective mucous membrane at the time of operation obviates the most severe pain and discomfort later. The patient eats breakfast the next morning. The time for the wound to cover over is lessened by half, and the amount of scarring is almost nil. No longer does tonsillectomy involve a large open wound set to catch food and become infected and so entail great discomfort and soreness for ten days. Rather it has become a question of how successful the surgeon has been in covering the most vulnerable and sensitive part of the wound and quickening the healing forces of nature to repair the throat with a minimum of discomfort, distortion and disturbance of function.

DESIGN FOR PLASTIC FLAPS (RETROTONSILLAR, SUPRATONSILLAR AND PRETONSILLAR INCISIONS)

The first step is freeing the plica retrotonsillaris.

An incision is made, starting at the base of the tongue at the lower pole of the tonsil and behind any infratonsillar lingual nodules which may be present. It is carried upward along the posterior attachments of the mucous membrane, which are freed little by

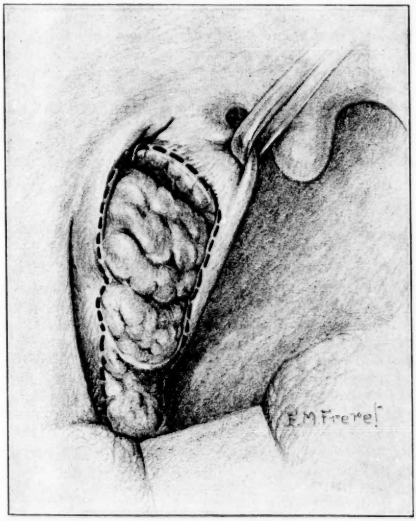


Fig. 2.—Extra incision at top frees anterior pillar from plica semilunaris, drawn taut by Allys clamp.

little where they meet and are attached to the surface of the tonsil. While this is being done the tonsil itself or the posterior pillar is held firm.

The meticulous saving of all mucous membrane for the whole length of the posterior pillar (down to the last eighth of an inch in width) is the aim of this procedure. Its accomplishment is much simplified by use

of a small curved knife set at an angle of 145 degrees which has been designed especially for this purpose.

The structure that is being freed from its attachment to the tonsil is the so-called plica retrotonsillaris. It is left at the end of the operation as a definite strip

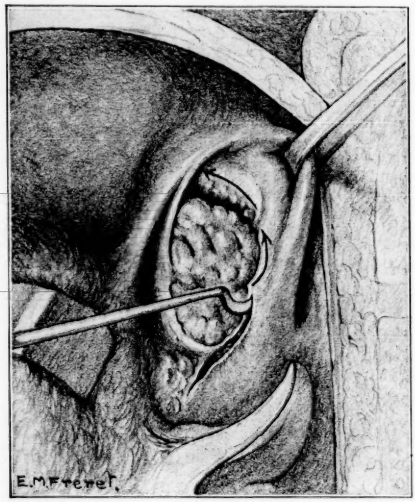


Fig. 3.—Incision up plica retrotonsillaris and plica semilunaris, with sickle knife, at a 145 degree angle.

of mucous membrane running straight or serrated the whole length of the posterior pillar.

The second step is freeing the plica semilunaris. When the incision starting from the base of the tongue has been carried upward past the lower lobe of the tonsil and past the hourglass constriction at the junction of the upper and the lower lobe and continued upward along the posterior attachment of the tonsil, freeing it from the posterior pillar (palatopharyngeus muscle), the cutting edge of the knife is directed forward along the edge of the plica semilunaris, which covers the upper pole of the tonsil. The blade is introduced under the mucous membrane and cuts "to the open." This sweep forward is continued as far as the edge of the anterior pillar (palatoglossus muscle), and at this point instead of going down, the incision turns upward and outward for a quarter of an inch. This significant extra incision when it joins the other incision frees the plastic flap from the anterior pillar. In this procedure lies the key to later success. In figure 3 is explained the theory of the three steps in the incision before they are united, and the important deviation, for ¼ inch at the top, from the classic horseshoe incision is shown as though it were a separate incision.

The third step is freeing the plica triangularis. The path of the knife is reversed; instead of following the edge of the anterior pillar above the tonsil it now retraces the quarter inch incision previously mentioned, and starting at the upper extremity of the plica triangularis the cutting edge of the knife is carried inward and downward, then slightly backward, following near the free edge of the plica triangularis and dissecting from the tonsil the greater part of the mucous membrane from the plica.

Fetterolf, in 1912, pointed out the ease with which the capsule (single layer of fascia) of the tonsil could be separated from the mucous membrane over the plica triangularis. This third part of the incision is based on the facts that he made so clear, and it is designed to recapture from the plica triangularis as much mucous membrane as can be saved (for plastic repair) without leaving any lymphoid tissue in the throat. The surgeon's judgment is called into play at the lower part of the plica triangularis to complete this incision near the base of the tongue short of, that is in front of, the infratonsillar nodules. In some cases it is necessary to bring the incision forward to avoid the lymphoid tissue that involves the surface between the lower pole and the base of the tongue.

A complete circumcision has thus been reached by the following steps: (1) incision up the posterior border, freeing the mucosa of the plica retrotonsillaris from its attachment to the tonsillar capsule; (2) incision forward to and up the edge of the anterior pillar along the free edge of the plica semilunaris, stripping the mucosa of the plica semilunaris from the capsule above the tonsil and from the muscle of the anterior pillar (palatoglossus), and (3) incision downward and inward near the free edge of the plica triangularis to the base of the tongue again in front of the infratonsillar nodules, stripping the mucous membrane of the plica triangularis from the capsule in front of the tonsil.

Each of these steps is designed to recapture all the available redundant mucous membrane that is free of lymphoid tissue. The combined effect of these

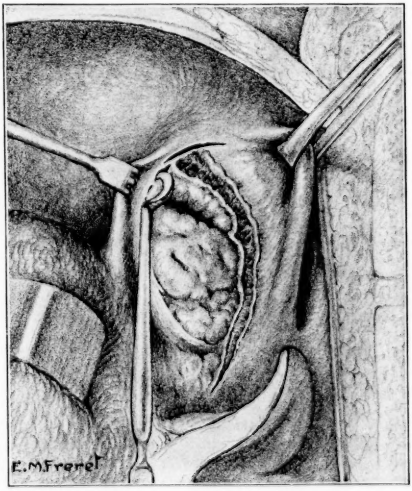


Fig. 4.—Incisions (1) retrotonsillaris and semilunaris (2) semilunaris from palatoglossus and (3) down free edge of plica triangularis.

incisions is designed (1) to leave a small loose flap of plica triangularis to cover in generously the edge of the anterior pillar and (2) to provide a much larger anchored flap including all the mucosa of the plica semilunaris and the plica retrotonsillaris for one major lapel to cover the posterior pillar. Finally, the incisions have separated these two flaps, the small for the ante-

rior and the large for the posterior pillar, freeing them from each other above and below.

A description of the various steps of operation between the incision and the final step of repair [1] is omitted from this paper except to note that after a dissection of the variety described there is left in the fossa a bundle of muscles which have been freed from the belt of the tonsil.

The bundle of muscle and fascia retracts upward and together with the vessels at the junction of the posterior pillar and the lateral wall forms the so-called bib of fascia and veins at the center of the fossa. It thus forms one of the elements used in repair; the other element is the flap of mucous membrane to which it will be joined.

After the removal of the tonsil, at the time at which the surgeon generally puts a ligature on the vessels at the center of the fossa, a new and simple step is introduced—the inclusion into the tie of the edge of the mucous membrane. This creates a situation of tension. It unites the mucous membrane from the plica semilunaris to a fold or "bib" of fascia at the center of the fossa. The little knot that holds them together must be applied so that the union of these two elastic elements is harmonious and the strain is slight.

The strain above and in front of the knot draws the summit of the fossa downward and backward, lessening the dead space, and at the same time and to the same degree the counterpull on the flap of mucous membrane draws it upward, outward and forward, so that as long as the tie holds it is an anchor point drawing toward it the upper part of the wound like a collapsing balloon

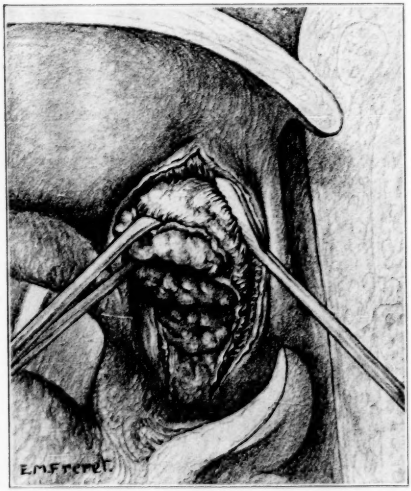


Fig. 5.—Undercutting major flap for posterior pillar, splitting layers of fascia.

and with an even counterpull drawing toward the same point the mucous membrane over the posterior pillar like a roller towel.

With one or two such well placed ties or sutures at the edge of a generous flap it is astonishing to see that in the first week the wound flattens out, collapses and covers itself with mucous membrane to the same extent that it would in three weeks if no ties were placed or if all the mucous membrane had been destroyed. These knots automatically disappear about the fifth day, leaving the flap firmly in place.

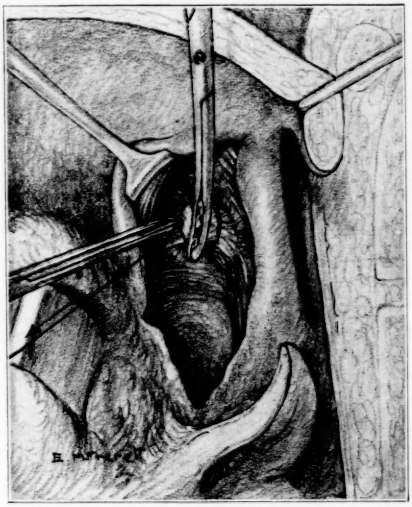


Fig. 6.—Catgut tie, main vessels, muscles and fascia at hilus.

In trying to account for this result it will be noted that there are a thousand invisible strands of pharyngeal fascia backed by the full, strong palatopharyngeus muscle attached to the hard palate above, which gives this pull its strength and creates a gliding flap which covers up the raw area with progressive steps from day to day.

REVIEW

Thus when the tonsil has been removed and any bleeding vessels tied (whether this is done under local anesthesia or general anesthesia makes no difference), a radically new step is introduced. The technic of this new step requires a study of detail. The mucosa at the edge of the posterior pillar is drawn gently toward the center of the fossa and is joined to fascia in the upper part by two ligatures of no. 1 catgut.

To retract the upper part of the wound, drawing the palatoglossus muscle at a level of the base of the uvula upward and outward with a Hurd retractor is a great help at this point, for this procedure narrows the fossa at the top and draws the mucosa over the edge of the posterior pillar not too far from its destined anchorage. There is a trick in the use of the Allys clamp at this stage. It seizes the edge of the mucous membrane and draws it upward and outward.

When the right position is reached one point of the Allys clamp is used to pin the mucous membrane in place while the clamp is slightly opened and allowed to engage the fascia. The trick lies in pressure on the first point of the clamp while it is open so that the mucosa does not slip away while the fold of muscle and fascia is being engaged by the other point. After this it is easy to apply a slipknot of no. 1 catgut just as in the usual technic in tying a vessel. The difference

1. Fowler, R. H.: Tonsil Surgery, Philadelphia, W. B. Davis Company, 1930, chapter 10.

is that in this case there is more material to tie down and the knot is pulled firm before the Allys clamp is removed and the catgut trimmed with scissors.

IMPORTANCE OF FASCIA IN THE TONSILLAR FOSSA

Investigation of the anatomy of the pharynx related to tonsillar surgery has brought out the importance of the pharyngeal fascia subjacent to the tonsil which is composed of the sheaths of the neighboring muscles, especially the palatopharyngeus, the palatoglossus, the superior constrictor and, to a lesser extent, the stylopharyngeus. Fascia is the undifferentiated tissue filling in the space between muscles, protecting the muscles and providing for their interplay. It is through the spaces of the fascia that the nerves, the veins and the arteries find their way. In fact, the tonsil operation is possible only because this strong, undifferentiated fibrous tissue can be separated into sheets. Anatomically considered, the removal of the tonsil is the problem of stripping away the mucosa (the surface of the throat) between the anterior and posterior pillars, lined as it is with lymphoid tissue, from the submucosa (pharyngeal fascia). In attempting to improve this operation it is of the highest importance to become familiar with the reconstruction of the fossa of the normal throat and to realize that the pharyngeal fascia of the fossa is attached above to the palate, to the styloid process and to the base of the skull. It stretches a network of fibrous tissue over the tonsillar fossa which not only forms the so-called capsule (the single layer that must be stripped away with the tonsil to hold it together) but also forms layers which may be left as a protective covering for the muscles of the fossa and the greatest single aid to rapid repair.

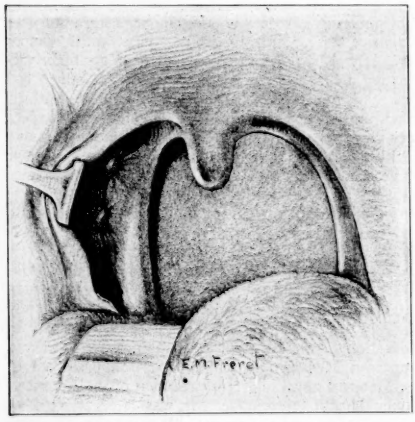


Fig. 7.—The major flap doubly knotted covers most sensitive part of posterior pillar.

On the other hand, when this pharyngeal fascia, instead of being left intact to give a sleek curved appearance in the fossa, is torn and harrowed by less sensitive and accurate dissections, the appearance of the fossa after the operation is changed from curved to angular lines. Once the network of fascia is torn, muscles of the pillars are revealed and exposed running at right angles to each other. The purpose of "muscle free dissection" of the tonsil is not only to leave the muscles of the fossa intact but to leave them covered with fascia.

The network of elastic tissue that is called fascia preserves the relationship of the muscles and insures their function six months after operation. When the fascia is left unbroken the muscles are unencumbered with scars and have their natural elasticity.

Fascia functions to support and give free play to muscles. Wherever it is damaged there is lessened function. Therefore it is of great surgical importance to preserve it.

COMMENT

Those who have learned to do a "muscle free dissection" and have learned to leave a fringe of mucous membrane the whole length of the posterior pillar will find plastic surgery of the tonsillar fossa fairly simple.

The posterior pillar flap is the size of a small finger nail. The successful operator can tie it in without a moment of added time when he is tying off the vessels. Perhaps it is better to give them the benefit of a tie of their own, so that the strain on the flap cannot complicate the closure of these vessels. The two elements fascia and mucous membrane are both elastic; when combined, they lend themselves to coordination and can be coaxed together with an Allys clamp. A knot of plain no. 1 catgut can then be used to unite them and thus anchor to a bit of jettisoned muscle a floating plastic flap which will cover progressively and protect the upper half of the exposed surface of the posterior pillar. It is then a simple matter to place a second tie to anchor the edge of the mucosa in exactly the same way, a quarter of an inch below the first, to the fascia at the center of the fossa, where there are large veins. Five days later the shrinkage of the wound causes the flap to cover in a greater area. Resorption and shrinkage of the muscle contract the wound at this stage of repair, and the floating flap by advancing restricts the area to be repaired by granulations (secondary intention).

As for the anterior pillar, it is well to leave as much of the mucosa from the plica triangularis as is free of lymphoid tissue to aid in the repair. This needs no ties or sutures. The saving of the mucous membrane at the time of the first incisions made during the operation makes the greatest difference to the comfort of the patient, even if no knots are used.

Study of the repair of the wound after a tonsillectomy shows that the most vulnerable and sensitive area is the exposed surface of the posterior pillar, for three reasons: its function in swallowing, its exposure to food and its neural supply from the glossopharyngeal nerve. From this fact it follows that the greatest help in assuaging pain and discomfort results from the plastic covering of this sensitive posterior pillar. Practically it works out that way. Some patients experience hardly any discomfort at all, eating breakfast without complaint the morning after the operation.

The difficulties of accomplishing this apparently simple procedure are numerous. In small children the available mucous membrane is on a miniature scale and is easily torn. In older persons there is plenty of material both in mucosa and well developed fascia. The greatest difficulty is getting a clear conception of the technic of cutting the flap for the posterior pillar to include the mucous membrane the whole length of the posterior pillar and all the plica semilunaris above the tonsil. The second difficulty is understanding the technic of placing the flap. The ties themselves are simple slipknot ties of no. 1 catgut which after five days take care of themselves.

The number of operations in which plastic flaps have been used has run into the thousands. There have not been any bad results—no necrosis and no destruction of the flap. The surgeon is warned to lift the flap upward a quarter of an inch as well as outward so as to avoid distortion by pulling down the palate. The idea is to replace the posterior pillar in its original form, and the flap should be drawn up toward the palate.

SUMMARY

Experiments with plastic surgery in the tonsillar fossa based on study of the anatomy give encouraging results. They tend to show that when the surgeon has carried out the conservation of tissues to a point at which all bundles of muscle originally attached to the tonsil are left in the throat and all the fascia of the throat is stripped from the tonsil except the last single intimate layer holding the lymphoid tissues together, when the tonsil has thus been shaved from the throat as is shown by the quilted appearance of its fascial surface, the surgeon has attained what may be called a "muscle free dissection." Then by the steps described, by conserving and placing all the mucous membrane originally attached to the tonsil, the healing of the wound is accelerated. A special incision separates the mucous membrane of the plica semilunaris and the plica triangularis above. This frees the plica semilunaris and the plica retrotonsillaris for a lapel that will cover a large part of the exposed surface of the posterior pillar.

The fascia of the fossa is drawn together and anchored to the edge of the mucosa, where in any case the larger vessels require a ligature (at the junction of the posterior pillar and the lateral wall). A second tie just below that serves to distribute further the tension on both fascia and mucous membrane.

It takes but a minute to cut a flap at the time of the first incision; it takes less than a minute to anchor this with a catgut slipknot to the fascia at the center of the wound in such a way as to help nature in the healing. There must be no sewing together of the two pillars. Each muscular pillar can be promptly protected with mucosa at the time of operation.

149 East Sixty-Second Street.

The Parallel Case of the House Rat.—About the middle of the thirteenth century, with the rise of modern civilization and the growth of world commerce, out of India came the black rat (Mus rattus) with long ears and tail and a glossy black coat. This species of rat had learned how to travel in sailing ships; it followed the beginnings of modern commerce over the whole world and as a migrant established itself successfully in nearly every country, where by "natural selection" it destroyed the local native species. Mus rattus thus became the "world rat." It held this position with increasing strength for five hundred years; then out of Asia early in the eighteenth century came the brown rat (Mus norvegicus), larger, more powerful and more vigorous than its predecessor the world-conquering black rat. Migrating with modern commerce, the brown rat made the practical conquest of the whole world in about one hundred and twenty-five years, practically succeeding the black rat in the title of "world rat." . . . In the rat world the record is not one of conquest by declared war and formal battle but one by the quiet immigration—a few at a time—of members of the invading species, which established itself, reproduced at a high rate, defeated its host-species in hundreds of "personal competitions" and finally succeeded to the ownership of the invaded territory. This, in short, is the biological formula, repeated many times over in the world of plants, animals and man, of conquest by immigration.—Laughlin, Harry H.: Immigration and Conquest: A Report of the Special Committee on Immigration and Naturalization of the Chamber of Commerce of the State of New York, May 15, 1939.

SCHOOL HEALTH POLICIES

CHARLES C. WILSON, M.D.
Director of Physical Education and Health Education, Board of Education
HARTFORD, CONN.

The term "school health policies" refers to the procedures that schools establish for protecting and promoting the health of school children. As physicians, regardless of the type of medical work in which we are engaged, we are concerned with these policies. If we are in private practice we are concerned because the children with whom the school is dealing are our patients. If we are in public health we are concerned because of needed coordination between school health education and public health activities, as well as because some communities place responsibilities for many school health activities on departments of health. Our interest in school health policies is particularly keen if we happen to be members of boards of education or are employed by boards of education as school medical advisors. If the former, we have the responsibility of deciding on policies for school health activities, and, if the latter, we must develop procedures in keeping with established policies. Further than this, as physicians we are interested in the health of all people, and we want to know if our schools are doing all they should to protect the health of school children, to give them guidance with health problems and to instruct them concerning the maintenance of health and ways in which they may be protected against disease.

As we study school health programs we are amazed to find the variations that exist. One city spends thousands of dollars on improving pupils' posture but conducts no tuberculosis case finding program, while a neighboring city does nothing about posture but does extensive testing with tuberculin. In a similar manner some schools spend considerable sums on smallpox vaccination and diphtheria immunization while others feel that schools should not be concerned with these matters. Health instruction in some schools is limited to elementary grade pupils, and the failure to develop high school health education means that pupils graduate with little or no scientific information concerning the maintenance of health and the prevention of disease. These extreme variations are the direct results of inadequate planning and the absence of clearcut statements of what schools should and should not do in the field of health.

A report entitled "Suggested School Health Policies" [1] is an attempt to remedy some of these confusing situations. This report, based on a preliminary study made by the Joint Committee on Health Problems in Education of the National Education Association and American Medical Association, was first presented to the Maternal and Child Health Section of the American Public Health Association. It was later referred to the following eight national organizations or committees:

1. American Association for Health, Physical Education and Recreation.

Read in the Fifth Symposium on Health Problems in Education at the Ninety-Second Annual Session of the American Medical Association, Cleveland, June 3, 1941.

1. "Suggested School Health Policies" was published in THE JOURNAL, April 27 and May 11, 1940, in the Journal of Health and Physical Education for May and June 1940 and in Hygeia for September, October, November and December 1940. Reprints are available from either the American Medical Association or the American Association for Health, Physical Education and Recreation.

2. American Academy of Pediatrics.

3. American Medical Association.

4. American Public Health Association.

5. American School Health Association.

6. Educational Policies Commission.

7. Joint Committee on Health Problems in Education of the National Education Association and American Medical Association.

8. National Organization for Public Health Nursing.

Each of these groups has been asked to study the preliminary report critically and to suggest modifications and additions for its future revision. Through this procedure the aim is to develop one set of school health policies which will be generally approved by educators, physicians and public health officials.

While it is impossible at this meeting to present the entire policies report, I would like to mention the eight fields of the school health program which it outlines and comment briefly on each.

1. *Provision of a Healthful Environment.*—The need for a healthful school environment is evident to all. The health and safety of school children is endangered by poorly lighted, poorly constructed school buildings or buildings with insanitary drinking fountains, inadequate washing facilities or unsatisfactory provisions for heating. There is need for periodic surveys of the sanitary facilities of school buildings and a program calling for at least an annual inspection of their sanitary condition. During recent years these questions of school sanitation have been given increased attention, but there are still many schools that do not come up to present day standards.

A healthful environment requires more than good sanitation; it requires that thought be given to the emotional tone of classrooms. This is part of the school's mental hygiene program, involving consideration of the health and personality of teachers, the type of teaching methods used, the forms of discipline, the methods of promotion, opportunities for expression of the pupils and the socializing effect of classroom activities. Only through consideration of the effect of these various procedures on the emotional development of pupils can a school be sure that it is providing a "healthful environment" as outlined in "Suggested School Health Policies."

2. *Care of Accidents and Sudden Sickness.*—Accidents are bound to occur at school and children will become sick at school. What should school personnel do in these instances? The policies report states that schools should have a "planned written program for the care of emergencies acknowledging school responsibility for (1) giving immediate care, (2) notifying parents, (3) getting pupils home and (4) guiding parents, where necessary, to sources of further treatment." It continues with the statement:

> Detailed instructions concerning the emergency care of various conditions, such as headache, cuts, bruises, dog-bites, suspected fractures, painful menstruation or pain in the abdomen, should be prepared by school physicians and copies distributed to each teacher. In general, it may be stated that ordinary first aid measures should be carried out in case of accidents but that the emergency treatment of sickness usually does not require that school personnel administer medication. The prescribing of acetylsalicylic acid for headache or pain, or the giving of sodium bicarbonate, epsom salt or essence of peppermint is unsound school policy and unsound health education. Most cases of early sickness will be helped best if the pupil is sent home and medication delayed until the physician makes a diagnosis and determines whether drugs are necessary. By following such a policy, schools will avoid the embarrassment caused by a pupil afflicted with a ruptured appendix because his parents postponed having a physician until they saw what effect the medicine given at school would have, and the equally embarrassing situation of giving acetylsalicylic acid for what turns out to be meningitis or brain tumor. In addition to avoiding embarrassment, the school will be doing good health education because it will be teaching that drugs are not taken for every condition which develops but usually only on the recommendation of one who understands their value, their limitations and their dangers.

3. *Prevention and Control of Communicable Diseases.*—There are many questions related to school policies in regard to the prevention and control of communicable diseases. Although legal responsibility for isolation, quarantine and other control measures are responsibilities of health departments, there are many things that schools can and should do. They should teach pupils and parents about preventive measures, the reasons for isolation and quarantine and the desirability of early care for those sick. They should encourage parents to keep sick children at home and should isolate and then send home those who become sick at school. In addition, they should, in cooperation with health departments, develop procedures to keep children who have had a communicable disease from returning to school while still in a communicable stage.

In regard to vaccination and diphtheria immunization, "Suggested School Health Policies" states:

> In each community there should be treatment facilities available for children of all economic levels. In most cases the school personnel will refer parents to their private physician for vaccination and for diphtheria immunization. Where parents cannot afford to pay for the services of a private physician, school personnel will refer children to hospital outpatient clinics, dispensaries, department of health clinics or other facilities which the community provides. As a general rule it is believed undesirable to conduct clinics in schools, although this may be the only way some communities will be able to solve the problem of making treatment available to all.

4. *Health Instruction.*—Health instruction is that part of the school health program which teaches pupils about the functioning of the human body, the causes and prevention of certain diseases and the acquirement and improvement of health and community health problems and programs. It is carried on by classroom teachers, teachers of home-making, biology, physical education and social studies.

"Suggested School Health Policies" contains many suggestions relating to health instruction, but perhaps of greatest interest is the paragraph which emphasizes the need of a scientific attitude toward health. This reads:

> Perhaps the greatest value of school health instruction comes from the development of a scientific attitude toward problems of health and disease and a realization that the principles of biology, physics and chemistry are as applicable to the human body as to matter or material elsewhere. It is believed a scientific attitude toward health and disease can break down superstitions and fads and thereby fit pupils to analyze critically the tremendous amount of misleading advertising presented in magazines and over the radio. Practice in distinguishing between fact and fallacy in health problems, learning where to find scientific data and authentic opinions, and studying various types of problems related to quackery and "patent

medicines" are methods to be employed in developing a scientific concept of personal and community health problems.

5. *Periodic Health Examinations.*—For some time many schools have given emphasis to periodic health examinations. In fact, this emphasis has been so great that many schools feel that they have a complete school health program when all they have is a plan for examinations. And, unfortunately, the emphasis on examinations has been placed on quantity rather than quality.

The report on policies recognizes the need for periodic examinations and suggests that "schools assume responsibility for developing a program of periodic health examinations, and that this program encourages examination of pupils by their private physician." It states further that "it will be necessary for schools to provide examinations for large numbers of pupils whose parents cannot or will not arrange examinations otherwise."

Periodic examinations should be supplemented by the "day by day observation of pupils and the referral for further investigation of pupils who do not seem well . . ."

In regard to the frequency of examinations, "Suggested School Health Policies" notes that there have been differences of opinion on this subject. The report states:

> Some advocate annual examinations while others favor three or four examinations during the school life of a child. Further study is needed before a scientific answer can be given. This study will have to consider the cost of examinations and the personnel needed and weigh these factors against the benefits that may accrue. In the opinion of this committee four examinations—two in the elementary school and two during the secondary school—should detect most abnormal conditions affecting growth, health and school progress as well as afford teachers information necessary for understanding pupils and adjusting school programs to their needs. Again, it is emphasized that periodic examinations should be supplemented by the day by day observation of pupils and arrangements whereby those who show evidence of disease or disorder may be examined and, where necessary, helped to secure needed treatment.

6. *Special Attention to Pupils Needing Medical or Dental Care.*—Saying that schools should give "special attention to pupils needing medical or dental care" is just another way of saying that examinations are not an end in themselves but should be followed by a program to help pupils secure needed treatment.

While emphasizing the importance of a follow-up program, the policies report expresses the view that treatment is not a function of schools. If existing treatment facilities for needy children are inadequate, it is suggested that the schools

> obtain specific data indicating the inadequacy . . . and present these data to those in charge of medical and dental relief programs with a plea that adequate facilities be provided. . . . Such a plan is superior to the establishment of treatment clinics in schools. If school clinics are developed they serve only the needs of children in the public schools, while clinics for adults and non-public school pupils may be operated under other administration. To avoid duplication or, at best, dual control it seems better that existing facilities for the medical and dental care of adults and children be increased if necessary so as to care for school children. Another reason for having treatment facilities at places other than the school is that treatment clinics in schools cause confusion as to the responsibility and function of the school health program. The presence of clinics at schools leads to the assumption that the school is responsible not only for health education and health supervision but for providing medical and dental care to all pupils. Education regarding the use of community facilities is a responsibility for the schools, but if, instead of doing this job, it provides treatment, it will not be teaching pupils and parents how to secure treatment but will be making them dependent on the school for these services. As stated by the White House Conference Report on the School Health Program, "Any policy that does for the individual what he can do for himself leaves him more dependent and less willing to care for himself when the protecting hand is withdrawn." Sound school health education teaches children and parents where and how to get treatment, so that when they leave school and when they are in need of care during vacations they will have learned how health needs may be met.

7. *Special Education Programs.*—The seventh field of school health activities refers to the adaptation of school programs because of physical limitations or the inauguration of special programs because of certain handicaps. In the first category would come those adaptations which are required in physical education because of cardiac conditions, convalescence, chronic fatigue or poor nutrition. And in the second category would be classes in lip reading for pupils with decidedly impaired hearing, sight-saving classes for the partially seeing and speech correction for those who stutter, lisp or have other impediments to clear speech. These special programs of education do much for handicapped pupils. They function best when the work of the special teacher or special school is closely coordinated with that of the school medical advisor.

8. *Supervision and In-Service Training for Teachers.*—The eighth, and last, part of the school health program as listed in "Suggested School Health Policies" recognizes the need for coordination of the health efforts of many persons within the school and for the coordination of school health efforts with the health activities of other groups in the community.

To quote from the report:

> All school personnel, namely janitors, classroom teachers, teachers of home economics, physical education, biologic sciences and social studies as well as the more strictly health trained people—as physicians, nurses, dentists, dental hygienists and dietitians—are concerned with activities of the school health program. To coordinate these groups so that the school health program will be a smoothly functioning unit without friction or duplication requires considerable planning, effort and diplomacy. Besides coordinating the various threads of health interest within the school, there is need of relating school health activities with the health interests of other groups, such as child welfare associations, departments of health, parent-teacher associations, medical and dental associations, and various clinics, hospitals, and dispensaries.

As stated previously, "Suggested School Health Policies" is a preliminary report. It is hoped that all individuals and groups concerned with or interested in school health programs will criticize the report, point out additional topics that should be included, suggest varying emphasis and in other ways help in the preparation of a better, more complete report. If you have not already done so, may I suggest that you secure a copy of this report and, further, that you plan to discuss its contents at your local medical meeting, with your superintendent of schools, your health officer and with others concerned with the health of school children.

249 High Street.

Clinical Notes, Suggestions and New Instruments

THE USE OF THE MILLER-ABBOTT TUBE IN SUBTOTAL COLECTOMY AND OTHER SURGICAL PROCEDURES

LELAND S. McKITTRICK, M.D., AND RICHARD WARREN, M.D.
BOSTON

The Miller-Abbott tube for the small intestine has acquired a permanent place in the therapeutic and diagnostic armamentarium of the surgical clinic. Its established uses are (1) in intestinal obstruction,[1] (2) in roentgen diagnosis of lesions of the small intestine[2] and (3) as an adjunct to operations involving intestinal suture to remove tension from the line of suture.[3] We have recently been impressed by its value in an additional group of conditions as a part of the technic of laparotomy. These cases are those in which it is of advantage to decrease the volume of the intraperitoneal contents (cases 1 and 2) or to have a method for the certain identification of intestinal loops (case 3). Its use in decreasing the intraperitoneal volume in operations for ventral hernia has been pointed out by Johnston.[4] Many others have undoubtedly employed the Miller-Abbott tube in these operations, but we have been unable to find further reports which emphasize this important use of it.

REPORT OF CASES

CASE 1.—A Jewish girl aged 16, unmarried, was admitted to the Massachusetts General Hospital Feb. 12, 1940 because of bloody stools of one year's duration and diarrhea for one month. A diagnosis of chronic ulcerative colitis was made by proctoscopy. The patient's course was stormy and febrile. She did not respond to therapy with barbiturates and responded only poorly to sodium sulfapyridine given intravenously. In spite of six transfusions she continued to lose ground, and ileostomy was performed on March 28. Her course after this was complicated by digestion and infection of the wound around the ileostomy, infections of the upper part of the respiratory tract and of the middle ear and hypoprothrombinemia. She was not able to leave the hospital until June 14.

She returned to the hospital on July 15 because of exacerbation of infection in the colon and acute arthritis of the left wrist and fingers and of the right knee and hip. She again ran a febrile course and became an extremely difficult problem in nutrition, but after ten weeks of preparation with high vitamin therapy, transfusions and a course of intravenous injections of sodium sulfapyridine a subtotal colectomy was attempted (L. S. McK.) on September 25. In spite of the fact that the patient had no clinical distention it was found at laparotomy that the small intestine was so dilated from her general atonia that adequate exposure to perform the operation safely could not be obtained. It was decided to close the abdomen and operate at a later date with the Miller-Abbott tube in place. This was done on November 19 (L. S. McK.), after the tube had been passing thirty-six hours and had appeared at the ileostomy. Only 3 feet (90 cm.) of tube was passed beyond the nostril to accomplish this. At operation the short segment of tube which lay within the small bowel had so emptied and threaded the intestine onto it that the operation was technically the easiest colectomy in the experience of the operator (fig. 1). Subsequent convalescence was uneventful.

CASE 2.—A married woman aged 39 was admitted to the hospital Nov. 20, 1940 because of menorrhagia for four months. A diagnosis of laceration of the cervix and chronic cervicitis was made in addition to that of uterine bleeding. After six days of study and preparation in the hospital a total hysterectomy was performed. Four days after operation the patient showed evidence of an infection of the wound with nonhemolytic streptococci and disruption of the wound. This could not be resutured because of the infection and was accordingly strapped together with adhesive tape, and a Miller-Abbott tube was immediately passed. This reached the lower part of the ileum in two days and prevented excess intraperitoneal pressure from causing recurrence of the evisceration. The wound healed by second intention.

Fig. 1.—Collapsed bowel threaded on tube; these three or four coils of bowel can easily be manipulated with a minimum of trauma.

CASE 3.—A Jewish girl aged 17, a college student, entered the hospital on Jan. 23, 1940. Seven months previously she had had an appendectomy with an uneventful convalescence and complete health until nine days before this admission, at which time she began to have crampy abdominal pain, fever and vomiting. Physical examination revealed a tender mass in the right lower quadrant of the abdomen. An operation was performed on the day of admission and drainage of an abdominal abscess done. The patient improved but continued to have signs of persistent infection in the region of the cecum, and a fecal fistula developed. The presumptive diagnosis was regional ileitis. All conservative means to close this over a period of ten weeks having failed, ileotransverse colostomy was resorted to on April 6.

A Miller-Abbott tube was passed on April 3, and roentgen studies on April 5 showed the tip of the tube to be about 1 foot (30 cm.) from the cecum (fig. 2). Between it and the cecum was a constricted area of intestine communicating with the fistula. At operation a mass of adhesions was found in the region of the cecum with several loops of intestine entering it. One of these was confidently identified as the end of the ileum

From the Surgical Service of the Massachusetts General Hospital.

1. Abbott, W. O., and Johnston, C. G.: A Nonsurgical Method of Treating, Localizing and Diagnosing the Nature of Obstructive Lesions, Surg., Gynec. & Obst. **66**: 691 (April) 1938. Leigh, O. C., Jr.; Nelson, J. A., and Swenson, P. C.: The Miller-Abbott Tube as an Adjunct to Surgery of Small Intestinal Obstruction, Ann. Surg. **111**: 186 (Feb.) 1940. McKittrick, L. S., and Sarris, S. P.: Acute Mechanical Obstruction of the Small Bowel: Its Diagnosis and Treatment, New England J. Med. **222**: 611 (April 11) 1940.

2. Boon, T. H.: Intubation of the Small Intestine: Demonstration and Localization of Partially Obstructive Lesions, Lancet **1**: 7 (Jan. 6) 1940. Abbott and Johnston.[1]

3. Ravdin, I. S., and Abbott, W. O.: The Use of the Miller-Abbott Tube in Facilitating One-Stage Resections of the Small and Large Bowel, Internat. Clin. **1**: 178 (March) 1940.

4. Johnston, C. G.: Decompression in the Treatment of Intestinal Obstruction, Surg., Gynec. & Obst. **70**: 365 (Feb., no 2A) 1940.

because of the presence of the tip of the tube within it. In the absence of the tube identification would have been impossible without extensive dissection into the infected area. Transection of the ileum and an ileotransverse colostomy were done on the identified loop. The tube was left down for five days after the operation. The patient had an uneventful convalescence, except for the development of an abscess beneath the scar left by her appendectomy, and all fecal drainage ceased. She was discharged in moderately good health on May 25 and returned to her home in another city. It was hoped that she might later become well enough to return for a colectomy on the right side. A few months later, however, it was reported to us that extension of her infection had developed with other complications and she had died.

COMMENT

The length of the human small intestine from the ligament of Treitz to the ileocecal valve, measured in situ in cadavers, averages 23 feet (700 cm.).[5] The length in the living person is impossible to measure accurately because it is continually changing. We do know, however, that because of tonus it is shorter than it is at the autopsy table.[6] The length of the mesenteric border is 13 feet 8 inches (420 cm.).[5] Probably a working value would lie somewhere between these two,

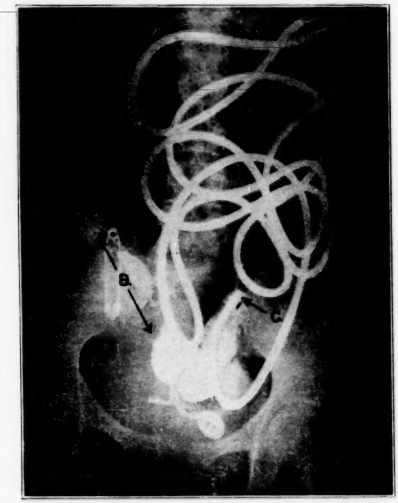


Fig. 2.—Appearance after injection of barium sulfate through the tube: *A*, displaced, deformed cecum; *B*, length of constricted, diseased terminal ileum; *C*, tip of tube, which is shown by the injected barium to be about 1 foot (30 cm.) from the ileocecal valve.

perhaps 19 feet (580 cm.). Since the length of tube generally necessary to pass through this amount of intestine is 3 to 4 feet (90 to 120 cm.) the amount of wrinkling, reefing and even intussusception that must take place with resultant decrease in volume of the peritoneal contents is considerable (fig. 1). The value of this procedure in giving easy exposure within the abdomen or in preventing or treating the disruption of the wounds becomes obvious.

The use of the Miller-Abbott tube in the identification of loops of the small intestine which cannot be identified by the operator because of adhesions is illustrated by case 3 and by several other cases in which we have given treatment. In order to be sure where the tip of the tube lies in relation to the cecum before operation a small amount of a weak suspension of barium may be injected through it and followed fluoroscopically until it enters the cecum (fig. 2). At laparotomy, then, the operator can identify the tip of the tube and know not only what part of the small intestine he is dealing with but also which direction from the part is upstream and which is downstream.

Cases in which the tube acts as a help in the manner indicated are not common, but when one has been encountered such help, in our experience, has been invaluable.

205 Beacon Street.

5. Monks, G. H.: Studies on the Surgical Anatomy of the Small Intestine and Its Mesentery, Ann. Surg. **42**: 543, 1905.

6. Van der Reis, V., and Schemhra, F. W.: Lange und Lage des Verdauungsrohres beim Lebenden, Ztschr. f. d. ges. exper. Med. **43**: 94, 1924.

MASSIVE HEMORRHAGE IN BENIGN PROSTATIC ENLARGEMENT

ROBERT LICH JR., M.D., LOUISVILLE, KY.,
AND
W. VINSON PIERCE, M.D., COVINGTON, KY.

It is our purpose in this paper to reemphasize the fact that uncontrollable prostatic hemorrhage can and does occur in benign hypertrophy. We have reference not to the hematuria so often associated with prostatism but indeed to a massive hemorrhage which actually threatens the life of the patient. That such a situation may occur spontaneously is significant, and its appreciation bespeaks caution in the use of instruments on the urethras of persons with prostatism.

Adler [1] in 1892 reported the first case of uncontrollable prostatic hemorrhage due to ruptured prostatic varices following urethral catheterization. Shivers [2] in 1935 reviewed the existing literature, and since that time 9 additional cases have been reported by Nitsche [3] and Hyman and Leiter,[4] bringing the total number to 35. Hyman,[5] since his article in 1938, has seen 2 additional cases, and we wish to report a case of our own.

ETIOLOGY

There are mentioned in the literature three primary causes of uncontrollable prostatic hemorrhage: (1) ruptured submucous varices of the hypertrophied prostate, (2) bleeding surface ulcerations of the gland and (3) generalized prostatic congestion with capillary bleeding.

Varices occur almost exclusively on the posterior commissure (median lobe). The formation of varices on this lobe was explained by Shivers. The veins emerge from the sides of the prostate and the posterior portion of the vesical outlet. The tonicity of the internal sphincter plus the pressure of the hypertrophied lateral lobes so impedes the venous return to the prostatic plexus that dilatation of the superficial veins results. These submucous vessels may rupture either spontaneously or through trauma.

Ulcerations may occur on the posterior commissure; however, their cause is often obscure. Infection might well be the most common cause, especially after instrumentation, though most authors have not discussed the degree or even the presence of infection. Lett [6] reported a case in which a stone in the bladder was the initiating factor in prostatic ulceration. Even trophic factors might well play a part in the production of apparently spontaneous ulcers of the prostate.

From the Urological Service of Dr. Owsley Grant, University of Louisville School of Medicine and the Louisville City Hospital.

1. Adler, J. M.: A Fatal Prostatic Hemorrhage, Berl. klin. Wchnschr., 1892, pp. 542-543.

2. Shivers, C. H. DeT.: Uncontrollable Hemorrhage from Benign Prostatic Enlargement: Report of a Case, J. Urol. **34**: 417-427 (Nov.) 1935.

3. Nitsche, P. H.: Uncontrollable Hemorrhage of Prostatic Enlargement, Am. J. Surg. **37**: 109-111 (July) 1937.

4. Hyman, Abraham, and Leiter, H. E.: Uncontrollable Prostatic Hemorrhage, J. Mount Sinai Hosp. **5**: 229-233 (Nov.-Dec.) 1938.

5. Hyman, Abraham: Personal communication to the authors.

6. Lett, Hugh: On Urinary Calculus, with Special Reference to Stone in Bladder, Brit. J. Urol. **8**: 205-232 (Sept.) 1936.

Generalized capillary bleeding may well be a secondary manifestation of Nitsche's factor of hypertension. Most physicians have seen a similar condition following transurethral prostatectomy done under spinal anesthesia. After the patient is returned to bed there develops from the surface of the freshly resected prostate a generalized venous oozing due to the gradually increasing blood pressure. Hence it is not hard to imagine that an analogous situation might occur in an enlarged congested prostate associated with considerable variable hypertension.

The actual cause of bleeding was given in only 16 of the 35 cases reported in the literature. In 6 cases, or 17.1 per cent, the bleeding was considered due to ruptured prostatic varices. In 3, or 8.5 per cent, it was due to ulcerations of the gland. Capillary oozing was considered the etiologic agent in 6 instances, or 17.1 per cent. In 1 instance an arteriosclerotic vessel was found at fault when the patient came to necropsy (Hyman and Leiter [4]).

In 9 cases, or 25.7 per cent, hemorrhage followed urethral instrumentation, and on 3 occasions, or 8.5 per cent, the hemorrhage followed suprapubic cystotomy and might well have been due to the trauma induced by the suprapubic catheter.

With the foregoing observations and facts in mind one may easily see why massive prostatic hemorrhage might occur, and its infrequency is indeed surprising. In fact, Shivers mentioned that he personally had been informed by men of wide urologic experience that they had never seen such a case. We have made an identical observation.

MANAGEMENT

In the treatment of patients with massive prostatic hemorrhage there are certain principles that must be observed. The use of a prostatic bag is undesirable as an agent for effecting hemostasis, since, if adequate tension is exerted on a bag to occlude the bleeding vessel, which may be concealed in a crevice created by the hypertrophied lobes of the prostate, there may be sufficient pressure to initiate separation of the prostatic lobes and thus enhance bleeding. Similarly, extremely tight intra-urethral packs can easily cause a mechanical separation of the prostatic lobes when the pack becomes soaked with blood and urine and expands. Hyman used in 1 case a thromboplastin-soaked pack to facilitate hemostasis.

Sutures must be used with extreme care and are practicable only when adequate exposure is possible. Tension cannot be exerted on sutures, since the adenomatous tissue is most friable, and if a suture pulls out additional bleeding will occur from the lacerated gland.

If the bleeding point is localized in an area of ulceration the cautery or coagulation electrode of the endotherm will often suffice. However, if there is bleeding from huge lacerated or ruptured varices or a generalized capillary hemorrhage, prostatic enucleation may be the only recourse.

Primary prostatic enucleation was undertaken 19 times, or 51.3 per cent, in all 37 cases (including the 2 additional cases of Hyman). Suprapubic cystotomy was found adequate to control hemorrhage in 8, or 21.6 per cent, of the cases in which operation was done. It is essential that the patient with massive hemorrhage of prostatic origin be treated actively. The fact that the patient is in general a poor risk is no contraindication for primary prostatic enucleation in the face of continued profound hemorrhage.

REPORT OF CASE

A white man aged 67 was admitted for acute urinary retention due to prostatism. The patient had had symptoms of prostatic enlargement for several months previously but had no other complaints. The blood pressure was 140 systolic and 70 diastolic, and no significant physical abnormalities presented themselves other than nonmalignant enlargement of the prostate, discovered by digital examination. Laboratory work showed no pathologic condition on microscopic, chemical or serologic investigations of the patient's blood and urine.

Cystoscopy was undertaken (by Dr. Lich), and, although the cystoscope was passed without effort, no visualization of either the bladder or the urethra was possible. When the telescope was removed there appeared a continuous flow of bright red clots and fresh blood. Visualization was again attempted, but without success. It was evident that a profound vesical hemorrhage had been initiated, and immediate surgical intervention was imperative.

Cystotomy was performed (by Dr. Pierce), and on the greatly enlarged posterior commissure there were found three huge submucous varices, the central one of which had ruptured and was actively bleeding. There was present also a submucous hematoma which extended over the entire superior surface of the posterior commissure. The hemorrhage was instantly controlled by using a proximally placed figure of eight suture to constrict the bleeding vessel. A lightly packed gauze tamponade was left in the posterior part of the urethra further to aid hemostasis. The patient enjoyed an uneventful course after both this procedure and the subsequent suprapubic prostatectomy.

SUMMARY

1. Massive prostatic hemorrhage, though rare, is a grave problem which must be handled with heroic surgical measures.

2. Ruptured varices and ulcerations, along with generalized capillary bleeding, constitute the chief etiologic factors in massive prostatic hemorrhage.

3. The case reported brings the total number in the literature to 38.

323 East Chestnut Street.

DEATH FROM PULMONARY EMBOLISM FOLLOWING THE INJECTION OF VARICOSE VEINS

LESLIE L. NUNN, M.D., AND JOHN H. HARRISON, M.D.
VANCOUVER, WASH.

If one considers the many cases in which varicose veins are treated by the injection of sclerosing solutions, the dreaded complication of pulmonary embolism is relatively uncommon. Pennoyer [1] in 1930 stated that only 4 cases had been reported up to that time; he stated further that in all these cases injection may have been done in the presence of acute or subacute phlebitis. Since 1930 2 more cases have been reported in this country and several have appeared in the foreign literature. Since 400,000 cases had been reported in which injection had been done, the possibilities of such a complication seem to be almost nil.

In 1928 McPheeters and Rice [2] reported 7 deaths in a series of 53,000 cases of injection therapy; Kettel [3] reported 20 deaths in a series of 60,000 cases, mortalities of 0.0025 per cent and 0.033 per cent respectively. These figures represent the combined mortality and include embolic accidents plus the various other causative factors of death.

Such low mortality figures may be due to the fact that this procedure carries practically no risk or that serious complications following injection therapy are overlooked or not reported. Kilbourne [4] has stated that "major complications following injection treatment of varicose veins are occurring with such frequency that the call of the voice of warning is imperative. It is unfortunate that such cases are concealed or divulged under the cloak of confidence. These accidents are preventable!"

Taylor [5] expressed the belief that the injection treatment is sufficiently hazardous to warrant ligation high in the saphenous vein prior to injection. In view of the extremely low mortality mentioned, such a measure seems a bit reactionary.

1. Pennoyer, G.: The Injection Treatment of Varicose Veins, Ann. Surg. **91:** 416 (March) 1930.

2. McPheeters, H. O., and Rice, C. O.: Varicose Veins: Complications, Direct and Associated, Following the Injection Treatment: A Review of the Literature, J. A. M. A. **91:** 1090 (Oct. 13) 1928.

3. Kettel, K.: Ueber Todesfälle im Anschluss an die Injektionsbehandlung von Varicen. Eine Literaturübersicht, Zentralbl. f. Chir. **58:** 1498 (June 13) 1931.

4. Kilbourne, N. J.: Varicose Veins: Indications and Contraindications to Injections, Ann. Surg. **93:** 691 (March) 1931.

5. Taylor, K. P. A.: Pulmonary Emboli Following the Injection of Varicose Veins, Am. J. Surg. **45:** 145 (July) 1939.

Smith[6] in reviewing a series of cases at the Mayo Clinic expressed the belief that emboli occur more frequently than the reported cases indicate. In 1939 in a series of 5,370 cases involving treatment by injection, he was able to demonstrate roentgenologically and clinically eight pulmonary infarctions, none of which proved fatal. Conditions which increase the hazard of pulmonary infarction in the injection treatment of varicosities are (1) acute or subacute phlebitis, (2) distal, concealed foci of infection, (3) senility and (4) the use of solutions which produce coagulation.

In the case we are reporting death occurred in the absence of any of the foregoing contraindications. An embolic accident followed the injection of an uncomplicated, asymptomatic, small varicose vein, the injection being done for purely cosmetic reasons. Autopsy confirmed the clinical impression of events leading to the patient's death.

REPORT OF CASE

Mrs. E. C., a housewife aged 41, entered St. Joseph's Hospital on Aug. 16, 1940 because of collapse, pain in the chest, cough and bloody sputum. Three days prior to entry she had been perfectly well and had been vacationing in the mountains. On August 14, two days prior to entry, the patient experienced a sense of malaise and chill which she attributed to a chilling received while swimming; the symptoms at that time were minimal, however, and after retiring she was quite comfortable until the morning of the following day, at which time she experienced a sudden stabbing pain in the right side of the chest anteriorly which radiated through to the back. The pain was aggravated by deep inspiration and by a nonproductive cough which developed shortly after the onset of the pain.

In spite of the discomfort associated with the cough, the patient remained up, walking about the camp at which she was staying; the pain and cough were somewhat less distressing toward evening. She was comfortable and able to rest that night. The following morning there was an exacerbation of symptoms, the pain was excruciating, the cough became productive of blood-tinged sputum and the patient became acutely ill. She was brought to the hospital from the camp in an automobile, a distance of 60 miles.

The previous history was essentially negative; there were no symptoms attributable to heart disease or pathologic conditions of the pelvis. Six weeks prior to the present illness, the patient had had an isolated saccular varicose vein in the right leg injected elsewhere. At the time of this injection the physician stated that there was no evidence of recent or old phlebitis. She received two injections of 2 cc. of a 5 per cent solution of sodium morrhuate with an interval of six days between injections. There was no decided reaction after this treatment, and a satisfactory thrombosis followed.

On physical examination the patient was acutely ill, pale and perspiring. She was coughing intermittently while lying flaccidly in bed and had pain in the right side of the chest. The skin was gray and clammy, there was no appreciable adenopathy, the eyes reacted properly and the ears, nose and throat were apparently normal. On entry the respiratory rate was 32; the respiratory movements were shallow and apparently equal on the two sides. There was diminution of resonance over the base of the right lung posteriorly, and the breath sounds were diminished anteriorly over the middle lobe of the right lung. The cardiac sounds were rapid and of fair quality; the pulse rate was 100, regular but of poor quality. The blood pressure on entry was 96 systolic and 60 diastolic. The abdomen was soft and relaxed, with no guarding or tenderness. There was an 8 by 3 cm. indurated area surrounding a thrombosed segment of the greater saphenous vein on the medial aspect of the right calf.

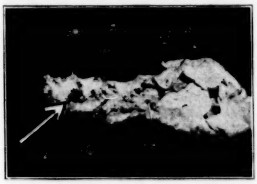
Laboratory data were as follows: The blood showed hemoglobin 122 per cent, erythrocytes 5,650,000 and leukocytes 26,600. The differential count of leukocytes showed polymorphonuclear neutrophils 81 per cent, small lymphocytes 15 per cent, large lymphocytes 1 per cent and monocytes 3 per cent. The sedimentation rate was within normal limits, the urine showed no sugar or albumin and microscopic examination gave negative results. The Kahn reaction was negative.

The clinical course was rapid; shortly after entry the blood pressure fell, the patient became semicomatose and, in spite of shock therapy and the administration of oxygen and large doses of opiates she became progressively worse and died twenty-four hours after entry. The clinical diagnosis was (1) chemical phlebitis of the right saphenous vein and (2) pulmonary embolism and infarction.

Autopsy was performed eight hours after death; the principal changes noted were as follows:

The right pleural cavity was free of adhesions and contained 500 cc. of clear, blood-tinged fluid; the right lung weighed 700 Gm. and the pulmonary tissue was well aerated except for a consolidated, red and hemorrhagic area 9 by 4 by 4 cm. extending out from the hilus. This infarcted area did not extend to the periphery of the lung, the pleural surface not being involved.

The pulmonary artery was opened in situ and a probe passed along the vessel until an obstruction was encountered. The heart and lungs were then removed *en masse* and the pulmonary artery and its branches opened. At the point of division of the right pulmonary artery into its main branches an embolus was seen; this was the site of obstruction encountered when the probe was inserted along the course of the vessel. The embolus occluded the lower of the two main branches of the right pulmonary artery and extended along the vessel for a distance of 5 cm. The embolus was dry and friable and adherent to the intima of the vessel; when it was removed some resistance was encountered and the intima at the site of attachment was roughened and covered with a thin layer of fibrin.



Appearance of a section of the saphenous vein showing the portion of the clot from which the embolus was released.

Over the medial surface of the left calf a thickened, indurated 11 cm. segment of the great saphenous vein was palpated. This section was dissected free and opened with some difficulty, as the walls were thickened and cut with abnormal resistance. The lumen of the vein was found to be narrowed but not completely occluded. The lumen of the vein transversing this segment was of varying caliber, the distal calf being narrowed almost to the point of occluding the vessel. At the midpoint of the segment a tributary vein joined the saphenous; above this the vessel was of much larger caliber. There was a thrombus occupying the distal portion of this dilated segment; the proximal end of the thrombus was ragged and frayed. The thrombus was stripped from the wall of the vein without difficulty.

Histologic examination of the thrombus taken from the saphenous vein showed it to be formed largely of platelets and fibrin; there were relatively few red cells. The embolus removed from the pulmonary artery consisted of fused platelets and fibrin and many red cells.

The pathologic diagnosis was (1) pulmonary embolism and infarction and (2) chemical phlebitis of the right great saphenous vein.

SUMMARY

Pulmonary embolism followed the injection of a small varicose vein of the leg, done solely for cosmetic reasons.

In a review of the literature on this subject it was found that following 110,000 injections the mortality rate was 0.0177 per cent. This figure represents all causative factors of death, including embolic accidents.

The incidence of embolism following the injection of ordinary varices in the leg is extremely small. However, we believe that it is of sufficient importance for the phenomenon to warrant attention.

301 East Tenth Street.

6. Smith, F. L.: Varicose Veins: Complications and Results of Treatment of Five Hundred Patients. Mil. Surgeon **85**: 514 (Dec.) 1939.

Special Article

GLANDULAR PHYSIOLOGY AND THERAPY

THE PHYSIOLOGY OF THE THYROID GLAND

JACOB LERMAN, M.D.
BOSTON

This special article is published under the auspices of the Council on Pharmacy and Chemistry. It is one of a series which will be published in book form as the second edition of "Glandular Physiology and Therapy." The opinions expressed in this article are those of the author and do not necessarily represent the views of the Council.—ED.

The thyroid is a gland of internal secretion whose chief function, so far as is known, is the elaboration and storage of its own peculiar hormone, namely, thyroglobulin, or the amino acid thyroxin contained therein. Originally it was a gland taking part in digestion. In the course of evolutionary changes, it has lost its connection with the alimentary tract. As Means [1] aptly said, "For a rôle in digestion it has substituted a rôle in growth and metabolism." To fulfil these functions, it is endowed with tremendous capacities for increasing or decreasing its activities, manifested by changes in size, blood supply, microscopic appearance and hormone content. In man the thyroid attains its relative maximum size just prior to puberty, corresponding to the time of maximum load on the organ from factors of growth and development. Simple colloid goiter, so common at this age, is the clinical manifestation of this physiologic fact.

HISTOPHYSIOLOGY OF THE THYROID

The gland is composed of a number of so-called follicles or acini, each one of which is a secretory unit. The average diameter, according to Wilson,[2] is 300 microns, but according to Jackson [3] it is somewhat smaller. The wall of the follicle consists of epithelium one cell deep. In normal glands the cell is about as high as it is wide (about 15 microns). The height of the epithelium has become a useful index of the functional activity of the thyroid.[4] The epithelium becomes flat when thyroid function is diminished and columnar when thyroid function is increased.

The cells of the follicle make the thyroid colloid; the lumen is the storehouse. The body taps this stored secretion as needed. Such a mechanism is obviously adapted to cope with great functional variations. The essential component of the follicle is the thyroid cell, which is fundamentally a secretory cell. Close study of this cell reveals certain characteristics, in addition to height, which betray its state of activity.

The importance of the mitochondria in relation to the functional activity of the cell was first noted by Goetsch.[5] Numerous experimenters have confirmed this. Cramer and Ludford [6] showed that as the cell passes from an inactive to a very active phase there occur heightening of the cell, diminution of colloid in the follicle and increase in the number and size of the mitochondria.

The Golgi apparatus is another cytoplasmic structure of physiologic significance. In the salamander Uhlenhuth [7] correlated the state of the Golgi apparatus to the secretory phases of the cell. In the guinea pig Krogh and Okkels [8] observed the changes after an injection of a thyrotropic extract of the anterior lobe of the pituitary. There occur, after two hours, hyperemia of the whole gland, diminution in the size of the follicles, disappearance of colloid and great hypertrophy of the Golgi apparatus. This is maintained for about seventeen hours. These histologic changes are paralleled by an increase in metabolic rate. Similar cytologic changes were observed by Severinghaus [9] in the thyroids of other species. The latter also noted that colloid droplets first appear in close proximity to the enlarged Golgi apparatus.

Oxidase granules were described by Okkels,[10] who related their distribution to thyroid function. In normal animals they are chiefly in the base of the cells. After injection of a thyrotropic extract they increase in numbers in the cells, invade the colloid and later become numerous in the interfollicular spaces as the colloid becomes vacuolated.

There is only one kind of cell in the follicle. It presents different appearances because it is observed in different stages of secretory activity. Recently Nonidez [11] described "parafollicular" cells, derived from parenchymal cells but occupying a position independent of the follicles. He suggested two possible functions for these cells: Either they represent a second type of secretory cell or they absorb something from the intrafollicular colloid and deliver it into the perifollicular blood vessels.

A good deal of controversy exists as to the direction in which the thyroid cells secrete—into the follicle or into the circulation. Means,[1] after going over the evidence thoroughly, favored the view that secretion can be in either direction. "Very likely, direct secretion into vessels occurs only when, in the face of great demand, the follicular storehouse has been emptied." Recently Algire,[12] employing a special method of transilluminating the thyroid gland of the living salamander, observed that, following injections of a thyrotropic anterior pituitary substance, there occur, in sequence, the gradual enlargement of a granule into a colloid droplet, the formation of new colloid droplets in proximity to the apical end of the nucleus, and the move-

From the Thyroid Clinic of the Massachusetts General Hospital.

1. Means, J. H.: The Thyroid and Its Diseases, Philadelphia, J. B. Lippincott Company, 1937.
2. Wilson, G. E.: The Thyroid Follicle in Man: Its Normal and Pathological Configuration, Anat. Rec. **37**: 31 (Nov.) 1927.
3. Jackson, J. L.: The Shape and Size of the Human Thyroid Follicle in Health and Disease, Anat. Rec. **48**: 219 (Feb.) 1931.
4. Rawson, R. W., and Starr, Paul: Direct Measurement of Height of Thyroid Epithelium; Method of Assay of Thyrotropic Substance; Clinical Application, Arch. Int. Med. **61**: 726 (May) 1938.
5. Goetsch, E.: Functional Significance of Mitochondria in Toxic Thyroid Adenomata, Bull. Johns Hopkins Hosp. **27**: 129 (May) 1916.
6. Cramer, W., and Ludford, R. J.: Cellular Activity and Cellular Structure as Studied in the Thyroid Gland, J. Physiol. **61**: 398 (June) 1926.
7. Uhlenhuth, Eduard: The Golgi Apparatus in the Thyroid Gland of Amphibians, in Its Relation to Excretion Polarity, Quart. J. Micro. Sc. **76**: 615, 1934.
8. Krogh, Marie, and Okkels, Harald: Sur l'histophysiologie du corps thyroïde. Stades initiaux de la sécrétion thyroidienne, Compt. rend. Soc. de biol. **112**: 1694 (May) 1933. Okkels, Harald: Studies on the Thyroid Gland: I. On the Histology and Cytology of Normal and Abnormal Thyroids in Man, Acta path. et microbiol. Scandinav. **9**: 1, 1932.
9. Severinghaus, Aura E.: Cytological Observations on Secretion in Normal and Activated Thyroids, Ztschr. f. Zellforsch. u. mikr. Anat. **19**: 653, 1933.
10. Okkels, Harald: Stades initiaux de la sécrétion thyroidienne. Les granulations oxydasiques, Compt. rend. Soc. de biol. **116**: 251 (May) 1934.
11. Nonidez, J. F.: Further Observations on Parafollicular Cells of Mammalian Thyroid, Anat. Rec. **53**: 339 (Aug.) 1932.
12. Algire, G. H.: The Study of the Living Thyroid Gland, Bull. School Med., Univ. Maryland **23**: 211 (April) 1939.

ment of colloid droplets from the apical end of the cell to the basal position, presumably for direct secretion into the vessels.

How the stored colloid gets into the circulation is also a moot question. It has been suggested [13] that it passes through intercellular channels. This view was opposed by Severinghaus,[9] who expressed the belief that the apexes of the cells push into the colloid in a pseudopodial-like manner, for the purpose of absorbing it. The colloid is then actively passed through the cells to the perifollicular vessels. The suggestion by Hertz [14] that endothelial leukocytes transport colloid from the follicle into the interfollicular spaces affords another explanation. The parafollicular cells of Nonidez, as indicated in a foregoing paragraph, may be engaged in a similar function. Recently Williams,[15] observing living thyroid follicles implanted in rabbits' ears, found that all follicles go through repeated cycles of activity. These cycles are divided into four stages—secretion, secretion and colloid release, partial collapse and recuperation. Secretion is toward the lumen. He found no evidence of active secretion across the base of the cell. In the second stage, when the follicle is filled with colloid, a droplet is pinched off and comes to lie within the follicular wall. The droplet slowly disappears, presumably by diffusion across the thin wall, but is never extruded into the interfollicular spaces. In other follicles a whole section of wall may become thin and colloid diffuse across it. No vacuoles are seen at the periphery of the colloid.

Since diffusion of a large molecule like thyroglobulin across a living semipermeable membrane is not likely, the presumption is that thyroglobulin is probably first digested (enzymatically) and that the resultant small fragments then diffuse through the follicle wall. This mechanism suggested by Williams, in contrast to the others, is consistent with my own observation [16] that thyroglobulin is not detectable in the peripheral blood or the blood of the thyroid vein of the normal person or of the patient with thyroid disease.

BLOOD SUPPLY AND INNERVATION OF THE THYROID

The blood supply of the thyroid is abundant. The blood volume of normal man (about 5 liters) moves through his thyroid about once an hour. There is a rich capillary network intimately connected with the parenchymal cells. Modell [17] recently described arteriovenous anastomoses and so-called "muscle cushions" in the thyroids of dogs. Williams,[15] on the other hand, was unable to see such structures in the living tissue. Their function is undoubtedly to regulate the flow of blood to the follicles and thus control the delivery of secretion to the body.

There is also a lymph plexus in close proximity to the follicles. A small portion of the thyroid's secretion leaves by this channel.

The thyroid is supplied by a rich network of autonomic nerve fibers, sympathetic from the cervical ganglions and parasympathetic from the vagus. It is still debated whether these fibers are secretory or vasomotor. The weight of evidence favors the view that they are not secretory and that secretory activity of the thyroid is governed indirectly by vasomotor control (Nonidez; [18] Cahane and Cahane; [19] Uotila [20]). On the other hand, there is good evidence that secretory activity of the pituitary can be influenced directly through nerve channels (Thomson and Collip; [21] Friedgood and Pincus [22]). This has been confused with direct nervous control of thyroid secretion. There is also evidence that the hypothalamohypophysial pathways may control thyroid function by their influence over the pituitary. Uotila [23] recently demonstrated that under normal circumstances section of the pituitary stalk does not affect the thyrotropic function; the thyroid consequently remains normal. However, under certain stresses, such as exposure to cold, animals in which the pituitary stalk has been sectioned fail to display the expected hypertropic changes in the thyroid.

THYROID HORMONE

One of the outstanding peculiarities of the thyroid is that it contains iodine in organic combination, a fact discovered by Baumann in 1896. The normal human thyroid contains about 0.186 per cent of iodine in terms of dried gland, the variation being 0.05 to 0.45 per cent (Gutman, Benedict, Baxter and Palmer [24]). Of the total iodine store in the body, the thyroid possesses about one third to one fourth. Seidell and Fenger [25] demonstrated a seasonal variation in the storage of iodine—lower in the early spring and higher in the late summer. Locality and food habits also play a role in determining the iodine content of the thyroid. Fenger [26] found iodine in fetal thyroids as early as the third month. It was demonstrated by Marine and Lenhart [27] that the iodine store varies inversely with the degree of hyperplasia.

Other structures of the body contain iodine, but the thyroid differs from them in its affinity for this element. It rapidly traps any extra iodine entering the circulation up to its maximum capacity, the amount trapped depending on the degree of thyroid activity (hormone depletion). This fact is the basis of the so-called iodine tolerance test in hyperthyroidism, introduced by

13. Hirschlerowa, Z.: Mikroskopisch-anatomische Untersuchungen an der Amphibienschilddrüse mit besonderer Berücksichtigung ihres Golgi-Apparates, Ztschr. f. Zellforsch. u. mikr. Anat. **6:** 234 (Aug.) 1927. Uhlenhuth, Eduard; Schwartzbach, S. S., and Thompson, G. P.: Die Physiologie der Thyreoaktivator bei Amphibien: II. Die Strukturveränderungen der Schilddrüse der mit Vorderlappen eingespritzten Salamander, Endokrinologie **16:** 9, 1935. McLendon, J. F.: The Release of Colloid from the Thyroid Gland by Centrifugal Force, Endocrinology **24:** 82 (Jan.) 1939.

14. Hertz, Saul, cited by Means.[1]

15. Williams, R. G.: Microscopic Studies of Living Thyroid Follicles Implanted in Transparent Chambers Installed in the Rabbit's Ear, Am. J. Anat. **62:** 1 (Nov.) 1937.

16. Lerman, Jacob: Iodine Components of the Blood: Circulating Thyroglobulin in Normal Persons and in Persons with Thyroid Disease, J. Clin. Investigation **19:** 555 (July) 1940.

17. Modell, Walter: Observations on the Structure of the Blood Vessels Within the Thyroid Gland of the Dog, Anat. Rec. **55:** 251 (Feb. 25) 1933.

18. Nonidez, J. F.: Innervation of the Thyroid Gland: III. Distribution and Termination of the Nerve Fibers in the Dog, Am. J. Anat. **57:** 135 (July) 1935; Nervous "Terminal Reticulum": Critique; Observations on Thyroid and Liver, Anat. Anz. **84:** 1 (March) 1937.

19. Cahane, Mares, and Cahane, Tatiana: Sur l'existence des centres nerveux infundibulaires réglant la fonction du corps thyroïde, Acta med. Scandinav. **94:** 320, 1938.

20. Uotila, U. U.: Rôle of Cervical Sympathetics in the Regulation of Thyroid and Thyrotropic Function, Endocrinology **25:** 63 (July) 1939.

21. Thomson, D. L., and Collip, J. B.: The Hormones, in Luck, J. M.: Annual Review of Biochemistry, Stanford University, Calif., Stanford University Press, 1933, vol. 2, p. 231.

22. Friedgood, H. B., and Pincus, Gregory: Studies on Conditions of Activity in Endocrine Organs: XXX. The Nervous Control of the Anterior Hypophysis as Indicated by Maturation of Ova and Ovulation After Stimulation of Cervical Sympathetics, Endocrinology **19:** 710 (Nov.-Dec.) 1935.

23. Uotila, U. U.: On the Rôle of the Pituitary Stalk in the Regulation of the Anterior Pituitary, with Special Reference to the Thyrotropic Hormone, Endocrinology **25:** 605 (Oct.) 1939.

24. Gutman, A. B.; Benedict, Ethel M.; Baxter, Blanch, and Palmer, W. W.: The Effect of Administration of Iodine on the Total Iodine, Inorganic Iodine, and Thyroxine Content of the Pathological Thyroid Gland, J. Biol. Chem. **97:** 303 (July) 1932.

25. Seidell, A., and Fenger, F.: Seasonal Variation in the Iodine Content of the Thyroid Gland, J. Biol. Chem. **13:** 517, 1913.

26. Fenger, F.: On the Iodine and Phosphorus Contents, Size and Physiological Activity of the Fetal Thyroid Gland, J. Biol. Chem. **14:** 397, 1913.

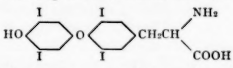
27. Marine, David, and Lenhart, C. H.: Further Observations on the Relation of Iodine to the Structure of the Thyroid Gland in the Sheep, Dog, Hog and Ox, Arch. Int. Med. **3:** 66 (Feb.) 1909.

Elmer.[28] Thyroid has the same affinity for iodine in vitro as in vivo (Marine and Feiss;[29] Rabinowitch and Frith[30]). Using radioactive iodine, Hertz, Roberts, Means and Evans[31] found that the normal rabbit's thyroid takes up iodine rapidly and becomes saturated within ten to fifteen minutes after an intravenous injection of iodine. Furthermore, certain types of hyperplastic glands are less able to utilize small amounts of iodine than are normal glands, although their capacity for taking up iodine from large doses is several times that of the normal gland. This may explain the need for relatively large amounts of iodine to induce a remission in the syndrome of exophthalmic goiter.

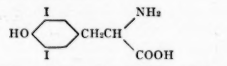
Preceding Baumann's discovery, it became apparent that the thyroid made a hormone. The syndrome associated with spontaneous atrophy of the thyroid, described by Gull[32] in 1874, was soon recognized as identical with the one due to total removal of the thyroid as noted by the Reverdins[33] in 1882 and by Kocher[34] in 1883. Knowledge of the curative value of thyroid substitution soon followed. In 1891 Murray[35] cured patients with hypothyroidism by subcutaneous injection of sheep's thyroid, and in 1892 Mackenzie[36] and also Fox[37] found that oral administration was equally effective.

Attempts at chemical isolation of the thyroid principle began with the discovery by Oswald[38] and by Hutchison[39] that the iodine of the thyroid is bound to a globulin (iodothyroglobulin) which is physiologically active and equal to whole thyroid. Other proteins of the thyroid do not contain iodine and are physiologically inert. It is the only protein present in colloid; it is also present in the follicle cells. The molecular weight of thyroglobulin was determined by Heidelberger and Svedberg[40] to be in the neighborhood of 700,000.

The early attempts at chemical purification of the colloid brought only partial success. Among others, Baumann, Müller and also Oswald obtained fractions of high iodine content and high physiologic potency by acid hydrolysis. In 1915 Kendall[41] isolated a pure crystalline substance containing 65 per cent iodine and possessing all the pharmacologic characteristics of whole thyroid. He named it "thyroxin." Harington[42] in 1926 demonstrated that Kendall's substance is an amino acid containing four iodine atoms—a hydroxyphenyl ether of tyrosine having the structural formula:



A year later Harington and Barger[43] synthesized thyroxin by conjugating two molecules of diiodotyrosine. The latter compound was isolated in pure chemical form from thyroid colloid by Harington and Randall.[44] It has the structural formula:



These two iodine-containing amino acids are the only ones, so far as present knowledge goes, to be found in the thyroid. Of the total iodine present in normal thyroid, about 30 per cent is in the form of thyroxin and about 70 per cent in the form of diiodotyrosine.

PHYSIOLOGIC POTENCY

What determines the physiologic potency of whole thyroid gland and how it is related to that of pure thyroxin are matters of physiologic interest. They are also of practical importance from the point of view of standardization of thyroid preparations. Several methods for measuring physiologic potency are available, for example, the Gudernatsch tadpole test and the Hunt acetonitrile test. However, the most convenient and most precise method of assaying thyroid for physiologic potency is by observing the effect on the rate of gaseous metabolism. In the past ten years my co-workers and I have assayed many and a variety of preparations, using as the index of activity the rate of increase of the basal metabolism of patients with full blown myxedema, from their low level, when the material is administered in daily doses for approximately two weeks. The basis for this technic has been discussed extensively by Means.[1]

Following the work of Kendall and of Harington on the isolation and nature of thyroxin, it was believed that thyroxin was the only active substance contained in thyroid and that measurement of the activity of a thyroid preparation could be obtained by measuring its thyroxin content. In other words, the calorigenic potency of a thyroid preparation could be better ascertained from its thyroxin content than from its total iodine content. Direct comparison between thyroxin and whole thyroid is impossible, because the former is absorbed irregularly from the gastrointestinal tract and the latter cannot be given parenterally. This difficulty was solved when Harington and Salter[45] isolated a polypeptide of thyroxin containing 50 per cent iodine, which Salter, Lerman and Means[46] found to be equally active whether given by mouth or intravenously. These investigators then found that thyroxin and the polypeptide of thyroxin are physiologically equal on the basis of their iodine content. On the other hand, the activity of whole thyroid containing a standard amount of thyroxin iodine is much greater than that of thyroxin

28. Elmer, A. W.: Iodine Tolerance Test for Thyroid Insufficiency, Endocrinology **18:** 487 (July-Aug.) 1934.

29. Marine, David, and Feiss, H. O.: The Absorption of Potassium Iodide by Perfused Thyroid Glands and Some of the Factors Modifying It, J. Pharmacol. & Exper. Therap. **7:** 557, 1915.

30. Rabinowitch, I. M., and Frith, A. B.: Iodine Studies: The Avidity of the Thyroid Gland for Various Iodine Compounds in Vitro, J. Clin. Investigation **1:** 473 (June) 1925.

31. Hertz, Saul; Roberts, A.; Means, J. H., and Evans, R. D.: Radioactive Iodine as an Indicator in Thyroid Physiology: Iodine Collection by Normal and Hyperplastic Thyroids in Rabbits, Am. J. Physiol. **128:** 565 (Feb.) 1940.

32. Gull, W. W.: On a Cretinoid State Supervening in Adult Life in Women, Tr. Clin. Soc. Lond. **7:** 180, 1874.

33. Reverdin, J.-L.: Accidents consécutifs à l'ablation totale du goitre, Rev. med. de la Suisse Romande **2:** 539 (Sept.) 1882.

34. Kocher, Theodore: Ueber Kropfextirpation und ihre Folgen, Arch. f. klin. Chir. **29:** 254, 1883.

35. Murray, G. R.: Note on the Treatment of Myxoedema by Hypodermic Injections of an Extract of the Thyroid Gland of a Sheep, Brit. M. J. **2:** 796 (Oct.) 1891.

36. Mackenzie, H. W. G.: A Case of Myxoedema Treated with Great Benefit by Feeding with Fresh Thyroid Glands, Brit. M. J. **2:** 940 (Oct.) 1892.

37. Fox, E. L.: A Case of Myxoedema Treated by Taking Extract of Thyroid by the Mouth, Brit. M. J. **2:** 941 (Oct.) 1892.

38. Oswald, A.: Die Eiweisskörper der Schilddrüse, Ztschr. f. physiol. Chem. **27:** 14, 1899.

39. Hutchison, R.: The Chemistry of the Thyroid Gland and the Nature of Its Active Constituent, J. Physiol. **20:** 474, 1896.

40. Heidelberger, Michael, and Svedberg, The: The Molecular Weight of Thyroglobulin, Science **80:** 414 (Nov. 2) 1934.

41. Kendall, E. C.: The Isolation in Crystalline Form of the Compound Containing Iodin Which Occurs in the Thyroid: Its Chemical Nature and Physiological Activity, Tr. A. Am. Physicians **30:** 420, 1915.

42. Harington, C. R.: Chemistry of Thyroxine: I. Isolation of Thyroxine from the Thyroid Gland, Biochem. J. **20:** 293, 1926; II. Constitution and Synthesis of Desiodo-Thyroxine, ibid. **20:** 300, 1926.

43. Harington, C. R., and Barger, G.: Chemistry of Thyroxine: III. Constitution and Synthesis of Thyroxine, Biochem. J. **21:** 169, 1927.

44. Harington, C. R., and Randall, S. S.: Observations on the Iodine-Containing Compounds of the Thyroid Gland: Isolation of dl-3: 5-di-Iodotyrosine, Biochem. J. **23:** 373, 1929.

45. Harington, C. R., and Salter, W. T.: The Isolation of l-Thyroxine from the Thyroid Gland by the Action of Proteolytic Enzymes, Biochem. J. **24:** 456, 1930.

46. Salter, W. T.; Lerman, Jacob, and Means, J. H.: The Calorigenic Action of Thyroxin Polypeptide, J. Clin. Investigation **12:** 327 (March) 1933.

polypeptide containing the same standard amount of thyroxin iodine (Means, Lerman and Salter [47]). Whole thyroid containing total organic iodine equal to standard thyroxin iodine gives a standard response. Moreover, Lerman and Salter,[48] observing the maintenance requirements of myxedematous patients on several brands of thyroid which contained different amounts of total iodine and of thyroxin iodine, concluded that total organic iodine, rather than thyroxin iodine, determines the calorigenic activity of whole thyroid.

Palmer and Leland,[49] however, using intact guinea pigs as test objects, came to the conclusion that thyroxin iodine, rather than total organic iodine, determines physiologic potency. Krogh and Lindberg,[50] using the same kind of test animal, came to similar conclusions. On the other hand, Meyer and Wertz,[51] using thyroidectomized rats, and White and collaborators,[52] using intact guinea pigs, have supported the contentions of Means, Lerman and Salter. In the past few years I have been comparing the effects of whole thyroid and thyroxin in normal and in thyroidectomized rabbits.[53] The results thus far emphasize the difficulty of using herbivorous animals in testing the effect of thyroid by mouth. In some instances the calorigenic response to thyroid by mouth equals that to thyroxin given intravenously in equi-iodine dosage; in others the calorigenic response to thyroid by mouth is much lower. These discrepancies are understandable when one realizes that stasis of food in the rabbit's stomach, frequently lasting eighteen to thirty-six hours, may result in destruction of ingested thyroglobulin.

The results obtained by Means and co-workers led to a puzzling paradox. The organic iodine of the thyroid is divided between thyroxin and diiodotyrosine. When separated, the latter is inert. Yet the activity of the whole thyroglobulin molecule is apparently due to all its iodine. It was suggested by Harington and Salter [45] that the high activity of whole thyroid iodine as compared with the thyroxin derived from it is due to such factors as chemical combination (peptide linkage) or optical activity of thyroxin. However, the polypeptide of thyroxin is not more potent than crystalline thyroxin in terms of iodine (Salter, Lerman and Means [46]), and l-thyroxin, which occurs naturally in thyroglobulin, is no more active than d-thyroxin (Salter, Lerman and Means [54]).

The synthesis of an active protein from the inert diiodotyrosine peptone by Salter and Lerman [55] may explain this paradox on the basis of Harington's theoretic concepts. According to Harington,[56] there is a special linkage in the thyroglobulin molecule between thyroxin and diiodotyrosine which is ruptured in the initial stage of isolation of thyroxin. He has visualized this linkage as follows: "It may be supposed that the iodine which reaches the thyroid is first introduced into the molecule of tyrosine to form 3,5-diiodotyrosine. The latter will then fulfil a dual role; part of it will be converted into thyroxine . . . and another part will be linked with the thyroxine so formed, together with other anino-acids, to form the true active principle of the gland."

PHYSIOLOGY OF THYROID HORMONE

Knowledge of the physiologic action of thyroid has been obtained by four different methods. The first method consists in observing the effects produced by destruction of the gland—by disease or by surgical ablation—in the experimental animal and in man. The second method consists in observing the effect of feeding the gland to subjects deprived of it. The third method consists in observing the effects of an excess of active substance, spontaneous or induced. The final method, and the most recent, consists in observing the effect on the thyroid of removal of other glands or of administration of other glands. Without a doubt, the thyroid hormone plays a vital role in the economy of the body. Its various functions are to be considered under several headings, but it must be emphasized that they are undoubtedly all interrelated.

Calorigenic Action.—Magnus-Levy first recognized that the thyroid plays a role in the regulation of heat production, or, its equivalent, oxygen consumption and carbon dioxide production. In other words, the rate of energy exchange, or metabolism, is regulated by the thyroid. This calorigenic action is most conveniently measured by determining the consumption of oxygen over a period of time. When the organism is deprived of all thyroid, the metabolism drops to about 40 to 45 per cent below normal in about 60 to 80 days. The rate of decline is predictable. It may be represented by the exponential equation $y = C + Ae^{-kt}$ which describes a decay curve.[57] When thyroid or thyroxin is administered, the metabolism rises, the rate of increase and level attained depending on the dosage used. In general the response to thyroid is inversely proportional to the initial level of metabolism—it is greater at low levels than at high levels for any given dose. At high environmental temperatures and humidity the effect of thyroxin is exaggerated (Schmidt and Schmidt [58]).

The means by which thyroid accelerates oxidative processes is not absolutely certain. It is not to be explained by increased muscular activity and is independent of adrenal activity (Aub, Bright and Uridil [59]). It involves every tissue of the body. Ample proof is available that the metabolism of tissue excised from thyroidectomized animals is low, and that of tissue excised from hyperthyroid animals, high. The experiments of Meyer, McTiernan and Aub [60] indicate that the calorigenic action is independent of the nervous system, since tissues denervated prior to administra-

47. Means, J. H.; Lerman, Jacob, and Salter, W. T.: The Rôle of Thyroxin Iodine and Total Organic Iodine in the Calorigenic Action of Whole Thyroid Gland, J. Clin. Investigation **12**: 683 (July) 1933.

48. Lerman, Jacob, and Salter, W. T.: Maintenance Requirements of Myxedema Patients: Clinical and Chemical Assay of Commercial Thyroid Preparations, J. Pharmacol. & Exper. Therap. **50**: 298 (March) 1934.

49. Palmer, W. W., and Leland, J. P.: Comparative Calorigenic Action of Normal and Pathological Thyroid Glands Administered in Equi-Thyroxine Doses, J. Clin. Investigation **14**: 619 (Sept.) 1935.

50. Krogh, Marie, and Lindberg, Anna-Louise: Studies on Thyroid Gland; Physiological Activity of Iodine in Thyroxin and in Normal and Pathological Thyroid Glands, Acta path. et microbiol. Scandinav. **9**: 21, 1932.

51. Meyer, A. E., and Wertz, Anne: The Calorigenic Efficiency of Thyroid Material in Relation to Thyroxine and to Iodine Content, Endocrinology **24**: 683 (May) 1939.

52. White, Julius; McGinty, D. A.; Anderson, L. P., and White, Florence R.: The Influence of Desiccated Thyroid, Thyroid Concentrate and Thyroxine on the Oxygen Consumption of the Guinea Pig, Endocrinology **24**: 693 (May) 1939.

53. Lerman, Jacob: Unpublished data.

54. Salter, W. T.; Lerman, Jacob, and Means, J. H.: The Calorigenic Action of D- and L-Thyroxin, J. Clin. Investigation **14**: 37 (Jan.) 1935.

55. Salter, W. T., and Lerman, Jacob: The Genesis of Thyroid Protein: Clinical Assays of Artificial Thyroid Protein in Human Myxedema, Endocrinology **20**: 801 (Nov.) 1936.

56. Harington, C. R.: Biochemical Basis of Thyroid Function, Lancet **1**: 1199 (May 25); 1261 (June 1) 1935.

57. Means, J. H., and Lerman, Jacob: The Curves of Thyroxine Decay in Myxedema and of Iodine Response in Thyrotoxicosis: Their Similarity and Its Possible Significance, Ann. Int. Med. **12**: 811 (Dec.) 1938. Boothby, W. M., and Baldes, E. J.: Activation and Decay Curves of Thyroxin, Proc. Staff Meet. Mayo Clin. **1**: 166, 1926.

58. Schmidt, L. H., and Schmidt, I. G.: The Relation of Environmental Temperature to the Action of Thyroxine, Endocrinology **23**: 553 (Nov.) 1938.

59. Aub, J. C.; Bright, E. M., and Uridil, J.: Studies upon the Mechanism of the Increased Metabolism in Hyperthyroidism, Am. J. Physiol. **61**: 300 (July) 1922.

60. Meyer, O. O.; McTiernan, C., and Aub, J. C.: The Effect of Thyroxin upon the Metabolism of Isolated Normal and Malignant Tissue, J. Clin. Investigation **12**: 723 (July) 1933.

tion of thyroid show elevated metabolism just as do tissues left intact. Moreover, it is not necessary that the animal be intact for the hormone to exert its characteristic effect. Canzanelli and Rapport [61] reviewed the results published on the effect of thyroid in vitro and reported that although thyroxin does not alter the oxygen consumption of tissues in vitro, thyroglobulin increases their metabolism considerably. The effect of diiodothyronine is inconstant and that of thyronine and that of diiodotyrosine negligible.

In its action the hormone, according to Zondek,[62] is in the nature of a catalyst, exerting its effect directly on the cell. Its action differs from that of an enzyme, which can accelerate reactions in an unorganized system. The hormone, to exert its effect, requires a living intact cell. On the other hand, Dye [63] concluded that an excess or a scarcity of thyroxin leads to an increase or a decrease in the amount, potency and effectiveness of the respiratory catalysts of cells, rather than that the thyroid hormone acts as an independent catalyst of itself. Such a role is also suggested by the observation of Scharles, Robb and Salter [64] that thyroxin produces a marked increase of liver amylase in the fasting animal.

Not only do thyroglobulin and thyroxin possess this calorigenic effect, but other substances, closely related to thyroxin, also have this property, but to a less extent. According to Harington,[56] any change in the molecule of thyroxin diminishes its physiologic activity. Thus Canzanelli and Rapport [65] found that in the dog tyrosine in large doses has a calorigenic effect, but this is only one two-thousandth of that of thyroxin. I have given large doses of tyrosine (1.5 Gm. daily, equivalent to the tyrosine content in 40 Gm. of thyroid protein) to 2 patients with myxedema without observing any metabolic change.[66] The former investigators also reported that diiodotyrosine is seven and a half times as active as tyrosine, diiodothyronine fifteen times as active as diiodotyrosine and thyroxin seventeen times as active as diiodothyronine. Thompson and co-workers [67] found some calorigenic action in diiodotyrosine when it was given to patients with myxedema, but only one ten-thousandth of that of thyroxin. Lerman and Salter [68] found that crystalline 3,5-diidothyronine and an iodothyronine-like substance derived from artificial iodoprotein each has an activity about one thirtieth to one fortieth of that of whole thyroid in terms of iodine.

Thus one must consider a molecule containing two atoms of iodine attached to a tyrosine nucleus as the minimum requirement for a substance having thyroxin-like physiologic properties. As indicated before, Harington [56] has claimed that in the body tyrosine is converted into diiodotyrosine, which is then elaborated into diiodothyronine and then into thyroxin. That such changes take place is suggested by the work of Salter and Lerman,[55] already referred to, on the formation of active protein from the inert diiodotyrosine peptone, which on redigestion yields "thyroxin-like" and "diiodotyrosine-like" peptones. The genesis of thyroid hormone may then be viewed as consisting of two processes: one, the building up of colloidal molecules from simpler polypeptide chains; the other, the combination of iodinated tyrosine residues to form iodinated thyronine residues. The mechanism of the second process is suggested by the work of Cohn, Salter and Ferry,[69] who showed that under certain conditions the phenolic groups of diiodotyrosine may undergo conjugation to form an iodized thyronine nucleus.

An important finding in this connection is that of Lerman and Salter,[68] Abelin [70] and Ludwig and von Mutzenbecher [71] that the simple process of iodinating protein by means of a gentle chemical reaction generates thyroid-like activity. It may be that simply the introduction of iodine into the tyrosine nucleus in the protein chain results in thyroid hormone. The significance of this is that the thyroid may not be the factory for the hormone but merely the storehouse for its distribution.

Action on Growth, Maturation and Differentiation of Tissue.—Congenital athyreosis in the human subject results in the dwarfism and juvenile habitus of the cretin. When thyroid is administered, growth is resumed. Conversely, a child suffering from thyrotoxicosis tends to be definitely taller than the average for the age. The failure of the cretin to attain the adult habitus is evidence that the thyroid plays a role in the maturation of the organism. In adult animals, according to Marine,[72] thyroidectomy produces little objective change, but in young ones thyroidectomy leads to stunted physical, mental and sexual development. This effect is more strikingly seen in the metamorphosing amphibia. Gudernatsch [73] showed that tadpoles metamorphose at an accelerated rate when thyroid is administered. Similar results have been obtained by Uhlenhuth [74] in the salamander. In both instances the animals mature to adulthood but do not change much in size.

The effect on the growth of individual tissues is seen in the retarded skin, hair and nail change in myxedema and in the delayed bony development in childhood athyreosis, i. e., delay in the appearance of ossification centers as well as in epiphysial union. In contrast, thyroxin stimulates growth of epiphysial cartilage in rabbits (Coryn [75]) and markedly accelerates the rate of eruption of incisor teeth in rats (Karnofsky and Cronkite [76]).

The growth-promoting action of the thyroid is, in all probability, a phase of its action on metabolic processes in general. According to Hammett,[77] the thyroid

61. Canzanelli, Attilio, and Rapport, David: The Effect of Thyroglobulin and Related Substances on the O_2 Consumption of Liver in Vitro, Endocrinology **21**: 779 (Nov.) 1937. Canzanelli, Attilio; Guild, Ruth, and Rapport, David: Further Observations on the Effect of Thyroglobulin and Thyroxin on the O_2 Consumption of Tissues in Vitro, ibid. **25**: 707 (Nov.) 1939.

62. Zondek, Hermann: The Diseases of the Endocrine Glands, translated by C. Prausnitz, ed. 3, Baltimore, William Wood & Company, 1935.

63. Dye, J. A.: The Action of Thyroxin on Tissue Respiration, Am. J. Physiol. **105**: 518 (Sept.) 1933.

64. Scharles, F. H.; Robb, P. D., and Salter, W. T.: Liver Amylase: The Effect of Nutrition and of Hormones, Am. J. Physiol. **111**: 130 (Feb.) 1935.

65. Canzanelli, Attilio, and Rapport, David: The Comparative Effects upon Metabolism of Intravenously Injected Tyrosine, Diiodotyrosine, Diiodothyronine and Thyroxine, Am. J. Physiol. **103**: 279 (Feb.) 1933.

66. Lerman, Jacob: Unpublished data.

67. Thompson, W. O.; Alper, J. M.; Thompson, Phebe K., and Dickie, Lois F. N.: The Effect of Diiodotyrosine on the Basal Metabolism in Myxedema, J. Clin. Investigation **13**: 29 (Jan.) 1934.

68. Lerman, Jacob, and Salter, W. T.: The Relief of Myxedema with Proteins of Extrathyroidal Origin, Endocrinology **25**: 712 (Nov.) 1939.

69. Cohn, E. J.; Salter, W. T., and Ferry, R. M.: The Amphoteric Properties of Globin and Iodized Globin, J. Biol. Chem. **123**: xxiv (May) 1938.

70. Abelin, I.: Nichtschilddrüsenstoffe mit Schilddrüsenwirkung: Weitere Erfahrungen über die Gewinnung schilddrüsenähnlich wirkender Substanzen aus künstlich jodiertem Eiweiss, Arch. f. exper. Path. u. Pharmakol. **181**: 250, 1936.

71. Ludwig, W., and von Mutzenbecher, P.: Ueber die Entstehung von Thyroxin durch Jodierung von Eiweiss, Ztschr. f. physiol. Chem. **244**: iv, 1936.

72. Marine, David: Physiology and Principal Interrelations of the Thyroid, J. A. M. A. **104**: 2250 (June 22) 1935.

73. Gudernatsch, J. F.: Fütterungsversuche an Amphibienlarven, Zentralbl. f. Physiol. **26**: 323 (June) 1912.

74. Uhlenhuth, Eduard: Relation Between Thyroid Gland, Metamorphosis, and Growth, J. Gen. Physiol. **1**: 473 (March) 1919.

75. Coryn, G.: Recherche expérimentale sur l'influence des glandes endocrines sur l'histologie du cartilage de conjugaison, Ann. d'anat. path. **16**: 27 (Jan.) 1939.

76. Karnofsky, D., and Cronkite, E. P.: Effects of Thyroxine on Eruption of Teeth in Newborn Rats, Proc. Soc. Exper. Biol. & Med. **40**: 368 (April) 1939.

77. Hammett, F. S.: Studies of the Thyroid Apparatus: XXIX. The Rôle of the Thyroid Apparatus in Growth, Am. J. Physiol. **76**: 69 (March) 1926.

hormone is concerned more with growth by increase in cell size than with growth by increase in cell number. It is essentially different from the growth hormone of the anterior lobe of the pituitary. However, there appears to be a synergism between the thyroid and the growth hormone of the anterior lobe of the pituitary. According to Evans, Simpson and Pencharz,[78] thyroid, which promotes normal growth of thyroidectomized animals, does not have this effect on hypophysectomized animals. Conversely, the growth produced by the growth hormone of the anterior lobe of the pituitary, although not dependent on the presence of the thyroid, is greater when the thyroid is intact. It is of interest that Sternheimer [79] obtained changes in liver glycogen, sugar and protein by the single injection of thyroxin which are consistent with those seen in growing tissues. These changes take place before the rise in oxygen consumption.

Action on the Distribution of the Water, Salts and Colloids of the Body.—When the organism is deprived of the thyroid, there takes place storage of water, salts and protein. In 1925 Boothby and associates [80] showed that in human myxedema a large amount of extra protein—so-called "deposit protein"—is stored in the body. It is contained in the body fluids and not in the cell protoplasm. When thyroid is administered, this extra deposit protein is quickly oxidized and eliminated in the urine along with the extra salts and water held in combination with the protein. Consequently, an appreciable diuresis is a characteristic finding when active hormone is administered to myxedematous patients for the first time. In fact, the diuretic action of thyroid may also be striking in normal persons, and is made use of in the treatment of nephrosis. The work of Byrom [81] indicates that the diuresis produced in patients with myxedema is accompanied by a loss chiefly of sodium salts, whereas in normal persons the loss is chiefly of potassium salts. Consequently, in the former the fluids are derived largely from extracellular sources whereas in the latter the fluids are derived largely from intracellular sources. This confirms Boothby's findings, mentioned earlier in this paragraph. Byrom has suggested that the abnormally collected protein in myxedema is in the nature of a mucoprotein derived from the ground substance of the cell. Since fetal tissue, like myxedematous tissue, contains an excess of mucin, it seems that one function of the thyroid is to provide the cells with a "mature" type of environment.

Soon after Boothby's work became known, Thompson [82] described a significant reduction in plasma volume in myxedema and a return to normal on treatment with thyroid. These results have been confirmed by Gibson and Harris,[83] who made the additional finding that the blood volume in thyrotoxicosis tends to be above normal. Along with the reduced plasma volume there is an increased concentration of plasma protein, with a corresponding increase in spinal fluid protein (Thompson and co-workers [84]). On administration of thyroid both the plasma and the spinal fluid revert to normal. In thyroidectomized animals the changes in the blood are similar to those in myxedema.

The metabolism of various inorganic salts is bound up with the state of thyroid activity. Aub and collaborators [85] showed that in hypothyroidism there is a diminished rate of exchange of calcium and phosphorus, the amounts eliminated in the urine and stool being less than in the normal person. The actual concentrations in the blood are not greatly altered. The reverse holds when thyroid is administered and in spontaneous hyperthyroidism. These effects, according to Low, Wilson and Aub,[86] are not brought about by changes in phosphatase activity in the bones. Talbot,[87] however, described low phosphatase in children with untreated hypothyroidism, associated with delayed osseous development. Thyroid treatment repairs these abnormalities. In the growth period the effect of thyroid on calcium metabolism may be different from that in later life. Thus Maroney and Johnston [88] found that retention of calcium and nitrogen was increased by the administration of thyroid to a cretin and to an adolescent after thyroidectomy. When administered in large doses thyroid may actually lead to premature cessation or retardation of growth (Smith and McLean [89]).

The metabolism of other inorganic salts is affected in the same manner as that of calcium and phosphorus. The blood levels of sodium and chloride in hypothyroidism are within normal limits,[90] but the urinary excretion of chlorides, according to Stephens [91] and Dr. Patricia Smith (personal communication), is low. These findings help differentiate myxedema due primarily to dysfunction of the thyroid from that due primarily to dysfunction of the pituitary.[92] In the latter, blood sodium and chloride are low and their excretion in the urine high (adrenal cortex insufficiency). The administration of thyroid restores the excretion of salt in primary hypothyroidism to normal, but in hypothyroidism secondary to pituitary dysfunction, by increasing the already excessive loss of salt, it may throw the patient into an addisonian crisis.

It should be indicated at this point that thyroid hormone also has an important control over blood lipoids. The blood cholesterol is elevated in hypothyroidism and decreased in hyperthyroidism. In fact, there is a close reciprocal relationship between basal metabolic rate and blood cholesterol.[93] On the experimental side Schmidt and Hughes [94] found that hyperthyroidism produces no change in cholesterol. Thyroidectomy, however, pro-

78. Evans, H. M.; Simpson, M. E., and Pencharz, R. I.: Relation Between the Growth Promoting Effects of the Pituitary and the Thyroid Hormone, Endocrinology **25**: 175 (Aug.) 1939.

79. Sternheimer, Richard: The Effect of a Single Injection of Thyroxin on Carbohydrates, Protein and Growth in the Rat Liver, Endocrinology **25**: 899 (Dec.) 1939.

80. Boothby, W. M.; Sandiford, I.; Sandiford, K., and Slosse, J.: The Effect of Thyroxin on the Respiratory and Nitrogenous Metabolism of Normal and Myxedematous Subjects: I. A Method of Studying the Reserve or Deposit Protein with a Preliminary Report of the Results Obtained, Tr. A. Am. Physicians **40**: 195, 1925.

81. Byrom, F. B.: The Nature of Myxedema, Clin. Sc. **1**: 273 (Nov.) 1934.

82. Thompson, W. O.: Studies in Blood Volume: The Blood Volume in Myxedema, with a Comparison of Plasma Volume Changes in Myxedema and Cardiac Edema, J. Clin. Investigation **2**: 477 (Aug.) 1926.

83. Gibson, J. G., Jr., and Harris, A. W.: Clinical Studies of the Blood Volume: V. Hyperthyroidism and Myxedema, J. Clin. Investigation **18**: 59 (Jan.) 1939.

84. Thompson, W. O.; Thompson, Phebe K.; Silveus, Esther, and Dailey, Mary E.: The Cerebrospinal Fluid in Myxedema, Arch. Int. Med. **44**: 368 (Sept.) 1929.

85. Aub, J. C.; Bauer, Walter; Ropes, M., and Heath, C.: The Relation of the Thyroid Gland to Calcium Metabolism, Tr. A. Am. Physicians **42**: 344, 1927.

86. Low, M. B.; Wilson, R. O., and Aub, J. C.: Phosphatase Activity of the Bones and Kidneys in Thyrotoxicosis, Proc. Soc. Exper. Biol. & Med. **31**: 447 (Jan.) 1934.

87. Talbot, N. B.: Influence of Thyroid Hormone on Serum Phosphatase, Endocrinology **24**: 872 (June) 1939.

88. Maroney, J. W., and Johnston, J. A.: Effect of Thyroid on Nitrogen and Calcium in the Growth Period, J. Pediat. **13**: 937 (Oct.) 1938.

89. Smith, E. E., and McLean, F. C.: Effect of Hyperthyroidism upon Growth and Chemical Composition of Bone, Endocrinology **23**: 546 (Nov.) 1938.

90. Lerman, Jacob: Unpublished data.

91. Stephens, D. J.: Chloride Excretion in Hypothyroidism, Proc. Soc. Exper. Biol. & Med. **43**: 742 (April) 1940.

92. Means, J. H.; Hertz, Saul, and Lerman, Jacob: The Pituitary Type of Myxedema: or Simmonds' Disease Masquerading as Myxedema, Tr. A. Am. Physicians **55**: 32, 1940.

93. Hurxthal, L. M., and Hunt, H. M.: Clinical Relationships of Blood Cholesterol, with a Summary of Our Present Knowledge of Cholesterol Metabolism, Ann. Int. Med. **9**: 717 (Dec.) 1935.

94. Schmidt, L. H., and Hughes, H. B.: The Free and Total Cholesterol Content of Whole Blood and Plasma as Related to Experimental Variations in Thyroid Activity, Endocrinology **22**: 474 (April) 1938.

duces a rise in which the normal ratio of free to esterified cholesterol is preserved. Thyroxin merely restores the normal level. In the rabbit I have been unable to find any change in blood cholesterol after complete thyroidectomy.[95] This finding may be due to the fact that the fat content of the rabbit's diet is low.

Action on Carbohydrate Metabolism.—Coggeshall and Greene[96] showed that thyroid feeding depletes hepatic glycogen even though the animals are on a diet high in carbohydrate. The conclusion was drawn that thyroid hormone not only depletes hepatic glycogen but also injures the liver so that it is unable to store glycogen. Conversely, thyroidectomy leads to excessive storage of glycogen in the liver. One frequently observes aggravation of latent or mild diabetes mellitus with the onset of hyperthyroidism. If myxedema and diabetes coexist, the relief of the myxedema intensifies the diabetes; on omission of thyroid the diabetes becomes milder. In fact, total thyroidectomy has been used in the treatment of diabetes mellitus.

Barnes[97] reported that feeding thyroid to the "Houssay dog" does not increase the mild glycosuria. He suggested that thyroid may exert its influence on carbohydrate metabolism through the pituitary which, in turn, may act on the adrenals. Similarly, thyroidectomy in pancreatectomized animals does not affect the diabetes in the way pituitarectomy (Houssay) or adrenalectomy (Long) does (Long;[98] Dohan and Lukens[99]). On the other hand, Soskin and co-workers[100] reported that the administration of thyroxin to hypophysectomized dogs helps maintain their normal blood sugar during periods of fasting, probably by increasing protein catabolism.

Action on the Nervous System.—The action of the thyroid on the nervous system is best observed in states of excess or of scarcity of the hormone. In the former case the person has emotional instability, increased irritability and sometimes gross disturbance in cerebration. In the latter case the person lives at a low emotional level, reacts sluggishly and cerebrates slowly; the memory is poor, and there is diminished sensory acuity. The effect of the thyroid on the vegetative nervous system is manifested by increase in vasomotor activity, peristaltic activity and activity of the sweat glands in thyroid intoxication and by the reverse of these phenomena in thyroid deprivation. An objective approach to evaluate this function of the thyroid was made by Ross and Schwab.[101] They found that the cerebral cortical alpha rate, as obtained by the electroencephalogram, is low in myxedema and returns to normal with the administration of thyroid. There is a good correlation between the alpha rate and the state of metabolism.

Action on the Muscular System.—In thyrotoxicosis there are changes in skeletal muscle which vary from mild myasthenia to advanced muscular atrophy, with corresponding degenerative changes in the muscle fibers. In hypothyroidism there is hypotonicity. Histologically, there is interstitial edema of muscle fibers. Spontaneous creatinuria is a characteristic finding in hyperthyroidism. According to Richardson and Shorr,[102] creatine tolerance is decreased in thyrotoxicosis, and as thyrotoxicosis declines creatine tolerance rises. Thorn[103] also found decreased creatine tolerance in hyperthyroidism and increased tolerance in myxedema. A useful correlation between total basal caloric output (a gage of the level of thyroid activity) and creatinine excretion (a gage of active muscle mass) has been obtained by Talbot, Worcester and Stewart,[104] confirming the earlier results of Palmer, Means and Gamble.[105]

Action on the Circulatory System.—The action of the thyroid on the circulation may be accounted for chiefly by its other actions, already discussed. A heightening in metabolism produced by thyroid calls forth an increased mass movement of blood. This is forthcoming from the more rapid heart action, the peripheral dilatation and the increased stroke volume of the heart. In hypothyroidism the reverse of these activities is observed. According to Zondek,[62] there is also a shift in the oxygen dissociation curve of the blood which facilitates delivery of oxygen to the tissues as metabolism rises. Increased cardiac irritability and tone in thyrotoxicosis and lowered tone in myxedema may be due to direct action of the thyroid hormone on the cardiac musculature. The cardiac enlargement so characteristic of myxedema is due to a combination of diminished cardiac tonus and myxedematous infiltration of the cardiac musculature. When thyroid is administered, edema disappears, tonus increases and heart size shrinks (Lerman, Clark and Means[106]).

Miscellaneous Actions of Thyroid Hormone.—Several actions of the thyroid are difficult of classification, but as knowledge improves their true significance should become clear.

Tolerance to some types of drugs is in some manner related to thyroid function. According to Benedict[107] (and my co-workers and I confirm it), patients with myxedema tolerate morphine poorly. It is our impression that such patients also tolerate digitalis poorly. On the other hand, patients with hyperthyroidism tolerate morphine and other sedative drugs extremely well. These findings may be attributed to the metabolic action of thyroid. In the same category is the acetonitrile reaction of Hunt[108]—the increased resistance of mice to acetonitrile, produced by the feeding of thyroid—and the increase in fluorine toxicity in animals receiving thyroid (Phillips, English and Hart;[109] Wilson and De Eds[110]).

In the field of animal husbandry, two types of speed-ups attributable to thyroid have been recently reported. The rate of egg laying by hens receiving thyroid is

95. Lerman, Jacob: Unpublished data.

96. Coggeshall, H. C., and Greene, J. A.: The Influence of Desiccated Thyroid Gland, Thyroxin, and Inorganic Iodine, upon the Storage of Glycogen in the Liver of the Albino Rat Under Controlled Conditions, Am. J. Physiol. **105:** 103 (July) 1933.

97. Barnes, B. O.: The Effects of the Endocrine Glands on the Carbohydrate Metabolism. A Working Hypothesis, Am. J. Physiol. **109:** 5 (July) 1934.

98. Long, C. N. H.: Recent Advances in Carbohydrate Metabolism with Particular Reference to Diabetes Mellitus, Ann. Int. Med. **9:** 166 (Aug.) 1935.

99. Dohan, F. C., and Lukens, F. D. W.: The Effect of Thyroidectomy upon Pancreatic Diabetes in the Cat, Am. J. Physiol. **122:** 367 (May) 1938.

100. Soskin, Samuel; Levine, R., and Heller, R. E.: Rôle of the Thyroid in the Carbohydrate Disturbance Which Follows Hypophysectomy, Am. J. Physiol. **125:** 220 (Feb.) 1939.

101. Ross, D. A., and Schwab, R. S.: The Cortical Alpha Rhythm in Thyroid Disorders, Endocrinology **25:** 75 (July) 1939.

102. Richardson, H. B., and Shorr, Ephraim: The Creatin Metabolism in Atypical Graves' Disease, Tr. A. Am. Physicians **50:** 156, 1935.

103. Thorn, G. W.: Creatine Studies in Thyroid Disorders, Endocrinology **20:** 628 (Sept.) 1936.

104. Talbot, N. B.; Worcester, Jane, and Stewart, Ann: New Creatinine Standard for Basal Metabolism and Its Clinical Application, Am. J. Dis. Child. **58:** 506 (Sept.) 1939.

105. Palmer, W. W.; Means, J. H., and Gamble, J. L.: Basal Metabolism and Creatinine Elimination, J. Biol. Chem. **19:** 239 (Oct.) 1914.

106. Lerman, Jacob; Clark, R. J., and Means, J. H.: The Heart in Myxedema. Electrocardiograms and Roentgen-Ray Measurements Before and After Therapy, Ann. Int. Med. **6:** 1251 (April) 1933; Further Observations on the Heart in Myxedema, ibid. **8:** 82 (July) 1934.

107. Benedict, Edward B.: Morphine in Myxedema, J. A. M. A. **94:** 1916 (June 14) 1930.

108. Hunt, R.: The Influence of Thyroid Feeding upon Poisoning by Acetonitrile, J. Biol. Chem. **1:** 33, 1905.

109. Phillips, P. H.; English, Honora, and Hart, E. B.: The Augmentation of the Toxicity of Fluorosis in the Chick by Feeding Desiccated Thyroid, J. Nutrition **10:** 399 (Oct.) 1935.

110. Wilson, R. H., and De Eds, F.: The Synergistic Action of Thyroid on Fluorine Toxicity, Endocrinology **26:** 851 (May) 1940.

increased above normal (Winchester[111]); also the production of milk and milk fat by cows is increased by thyroid (Graham[112]) and reduced by thyroidectomy.

Althausen and Stockholm[113] have shown that absorption from the gastrointestinal tract is regulated by the level of thyroid activity. Thus the absorption of various sugars is slow in thyroidectomized animals and rapid in hyperthyroid ones. In the light of these results dextrose tolerance tests in patients with myxedema and in patients with exophthalmic goiter acquire new significance.

The thyroid controls the blood-forming organs in a manner that is not clearly understood. In hyperthyroidism an excess of mononuclear cells in the blood stream is a frequent finding (Hertz and Lerman[114]); in myxedema moderate anemia is common (Lerman and Means[115]).

BLOOD IODINE

Of the normal blood iodine of about 8 to 10 micrograms per hundred cubic centimeters of blood, a small portion is due to circulating inorganic iodine, representing exogenous material; the remainder is due to organic iodine and may represent the amount of circulating hormone. The concentration of iodine in the blood fluctuates. Diet, exercise, medication and season are among the factors influencing the level of blood iodine (Salter[116]). The level fluctuates with thyroid function—it is lowered in myxedema and raised in thyrotoxicosis.

The nature of the organic iodine in the blood has been a puzzle. Lunde, Closs and Pedersen[117] separated total iodine into alcohol-soluble and alcohol-insoluble fractions. According to Salter,[116] such separations are artificial. Recent experiments indicate that most of the iodine is bound to protein but can be extracted from the protein by butyl alcohol (Trevorrow[118]). This extract, in turn, can be separated into "thyroxin-like" and "diiodotyrosine-like" fractions. Salter has suggested the following classification of iodine fractions in the plasma: (1) "I" iodine, nonprecipitable and presumably inorganic, and (2) "P" iodine, the maximum amount precipitated with the protein. The "P" fraction may then be separated into a "T" (thyroxin-like) fraction and a "D" (diiodotyrosine-like) fraction. Indeed, Trevorrow[118] found the ratio of "T" to "D" blood iodine the same as the ratio of these fractions in human thyroids.

That the blood contains circulating hormone is indicated by the fact that the oxygen consumption of mouse liver is increased by the addition of blood from patients with exophthalmic goiter and decreased by blood from myxedematous patients as compared with normal blood (Salter and Craig[119]). The failure of crystalline thyroxin to increase the consumption of oxygen is presumptive evidence that the circulating hormone is not the same as thyroxin. Canzanelli and Rapport[61] also found that thyroxin does not increase the metabolism of tissues in vitro, whereas thyroglobulin does. The phenomena observed by Salter and Craig are "thyroglobulin-like" rather than "thyroxin-like." In addition Perkin has been unable to recover thyroxin from the organic iodine fraction in his method of separating organic and inorganic iodine in the blood.[120] He is able to recover injected crystalline thyroxin from the organic fraction by the same technic after a lapse of three days. He concludes that the "thyroxin-like" fraction in the blood is not the same as crystalline thyroxin.[121]

As indicated, I^{16} have been unable to detect any thyroglobulin in the serum of normal persons or of patients with myxedema or hyperthyroidism. Consequently, it seems unlikely that intact thyroglobulin is represented in the organic iodine fraction.

RELATION OF THE THYROID TO OTHER ENDOCRINE ORGANS

It is obvious that in a complex mechanism like the body the thyroid is only one link in the elaborate coordinating system of glands. It acts on its endocrine partners and is in turn acted on by them, particularly by the central gland—the pituitary. The thyroid may influence other endocrine glands in several ways: (1) by a specific influence on all endocrine glands, (2) by a specific influence on the pituitary, (3) by a calorigenic effect on all endocrine glands and (4) by a calorigenic effect on the pituitary.

The effect of the thyroid on the pituitary is especially significant. Through this mechanism the thyroid indirectly controls the function of all glands and organs regulated by the pituitary. The thyroid, in turn, is controlled by the secretions of other glands or organ metabolites by way of the blood stream or by way of the nervous system. It is of interest that Foot, Baker and Carrel,[122] cultivating human thyroids in toto in the Lindbergh apparatus, showed that the histologic picture reached by the gland seems determined not by the phase of activity which existed while it resided in the body but by the nature of the perfusate used during its existence in vitro. In other words, it seems that humoral factors reaching the gland are more important than the condition inherent in the gland.

Pituitary.—Inasmuch as the pituitary-thyroid relationship is discussed fully in another chapter, I shall merely touch on the high lights. Knowledge of this relationship probably began with the observation of Rogowitsch[123] that in rabbits total thyroidectomy causes pituitary hypertrophy. It was shown by Houssay and associates[124] that the compensatory hyperplasia following subtotal thyroidectomy does not take place in the absence of the hypophysis. Moreover, according to the recent review by Means,[125] there is more thyrotropic activity in the blood and urine of

111. Winchester, C. F.: Influence of Thyroid on Egg Production, Endocrinology **24**: 697 (May) 1939.

112. Graham, W. R., Jr.: The Effect of Thyroidectomy and Thyroid Feeding on the Milk Secretion and Milk Fat Production of Cows, J. Nutrition **7**: 407 (April) 1934.

113. Althausen, T. L., and Stockholm, M.: Influence of the Thyroid Gland on Absorption in the Digestive Tract, Am. J. Physiol. **123**: 577 (Sept.) 1938.

114. Hertz, Saul, and Lerman, Jacob: The Blood Picture in Exophthalmic Goitre and Its Changes Resulting from Iodine and Operation: A Study by Means of Supravital Technique, J. Clin. Investigation **11**: 1179 (Nov.) 1932.

115. Lerman, Jacob, and Means, J. H.: Treatment of the Anemia of Myxedema, Endocrinology **16**: 533 (Sept.-Oct.) 1932.

116. Salter, W. T.: Iodine Metabolism or Fluctuations in Body Iodine, Physiol. Rev. **20**: 345 (July) 1940.

117. Lunde, G.; Closs, K., and Pedersen, O. C.: Untersuchungen über den Jodstoffwechsel: III. Untersuchungen über den Blutjodspiegel bei der primären Thyreotoxikose, Biochem. Ztschr. **206**: 261, 1929.

118. Trevorrow, V.: Studies on the Nature of the Iodine in Blood, J. Biol. Chem. **127**: 737 (March) 1939.

119. Salter, W. T., and Craig, F. N.: Vicarious Metabolic Response: The Oxygen Consumption of Surviving Tissues in Plasma from Hyperthyroid Organisms, J. Clin. Investigation **17**: 502 (July) 1938.

120. Perkin, H. J., and Hurxthal, L. M.: The Fractionation of the Iodine of the Blood in Thyroid Disease, J. Clin. Investigation **18**: 733 (Nov.) 1939.

121. Perkin, H. J.: Personal communication to the author.

122. Foot, N. C.; Baker, L. E., and Carrel, Alexis: The Behavior of Abnormal Human Thyroid Tissue Cultivated in the Lindbergh Apparatus, J. Exper. Med. **70**: 39 (July) 1939.

123. Rogowitsch, N.: Die Veränderungen der Hypophyse nach Entfernung der Schilddrüse, Beitr. z. path. Anat. u. z. allg. Path. **4**: 453, 1888.

124. Houssay, B. A.; Biasotti, A., and Magdalena, A.: Hypophyse et thyroide. Hypophyse et hypertrophie compensatrice de la thyroide, Compt. rend. Soc. de biol. **120**: 142 (May) 1932.

125. Means, J. H.: Diseases of the Thyroid Gland, New England J. Med. **221**: 820 (Nov. 23) 1939.

myxedematous patients and patients who have undergone thyroidectomy, and less in the blood and urine of thyrotoxic patients before operation, than in the blood and urine of normal persons.

There is considerable experimental evidence that the thyroid hormone exercises a depressing effect on the thyrotropic function of the anterior lobe of the pituitary (Aron and co-workers;[126] Kuschinsky;[127] Marine;[72] Uotila[128]). Uotila found that thyroxin produces atrophy of the thyroid to the same degree that hypophysectomy does and attributed the atrophy to the depressing effect of thyroxin on the anterior lobe of the pituitary. It is of interest in this respect that Belasco[129] observed a diminution in the consumption of oxygen by thyroid tissue excised from animals previously given injections of thyroxin.

Conversely, numerous investigators have shown that pituitarectomy results in involution of the thyroid. The syndrome described by Simmonds in 1914 represents the equivalent in man of total hypophysectomy. The essential symptoms are progressive wasting, weakness, subnormal metabolism, hypotension, hypoglycemia and cessation of sexual activity. Pathologically, there is hypoplasia of all the endocrine glands. In most instances the picture of full blown athyreosis is not present. In a few cases, however, as recently described by Means, Hertz and Lerman,[92] the dominant feature is myxedema, i. e., pituitary myxedema. These patients require treatment by replacement with preparations of the various tropic substances in proper combination. Administration of thyrotropic extract alone (Bulger and Barr[130]) or of thyroid alone (Means, Hertz and Lerman[92]) will aggravate the condition due to adrenal cortex insufficiency and result in death. Thyroid treatment should be accompanied by therapy aimed to protect the adrenals, namely, the administration of large amounts of salts, anterior pituitary adrenotropic extract or extract of adrenal cortex, and gonadotropic extracts or androgen or estrogen.

The experiments of P. E. Smith, B. M. Allen and others have emphasized the role of the anterior lobe of the pituitary as the regulator of the thyroid. That the thyrotropic principle of the anterior lobe of the pituitary regularly causes thyroid hyperplasia, increased metabolism and other evidences of increased thyroid function is now well established. This action is probably directly on the thyroid cells, because isolated thyroid tissue may be stimulated to increased metabolism by an extract of anterior lobe containing the thyrotropic factor. Williams[15] observed that the secretory cycle of thyroid follicles implanted in the rabbit's ear is accelerated by giving the animal thyrotropic material.

The relation of the posterior lobe to the thyroid is less clear than that of the anterior lobe. Mahoney and Sheehan[131] produced experimental diabetes insipidus in dogs by placing a clip on the pituitary stalk, and were then able to abolish the diabetes by total thyroidectomy. Feeding thyroid caused a return of the polyuria, which could then be abolished by injecting a solution of posterior pituitary. Apparently the diuretic action of thyroid may be antagonized by the antidiuretic action of the posterior lobe of the pituitary. In the rat, however, Swann and Johnson[132] found little influence by the thyroid on the diabetes insipidus following removal of the posterior lobe of the pituitary. Thyroid administration does not affect the rat's fluid exchange.

Adrenal.—In Addison's disease there is often a moderate drop in basal metabolism. This is not due to any gross disturbance in the function of the thyroid, nor is there any histologic disturbance found in the thyroid at autopsy. In the experimental animal, on the other hand, Marine and Baumann[133] and Davis and Hastings[134] found that removal of the adrenals causes a rise in metabolism. Again, as Means, Hertz and Lerman[92] emphasized, patients with hypofunction of the adrenal cortex, be it primary or secondary to pituitary disease, may be thrown into a crisis of adrenal failure by administration of thyroid. Zondek[62] stated that thyroidectomized animals survive total adrenalectomy longer than those possessing thyroids. Thyroid administration produces enlargement of the adrenal cortex (Uotila;[128] Ingle and Higgins[135]). Similarly Hoen, Langefeld and Oehme[136] showed that a large dose of desoxycorticosterone administered to guinea pigs antagonizes the rise in metabolism caused by thyroxin and also hinders the hypertrophy of the adrenal cortex seen in induced hyperthyroidism. On the other hand, Bock[137] showed that an extract of adrenal cortex plus thyroxin accelerates metamorphosis of tadpoles and axolotls at a greater rate than thyroxin alone. The administration of salt to hypophysectomized rats, according to Evans and co-workers,[138] produces a rise in their reduced metabolism, whereas the injection of salt into normal animals reduces their metabolism. Interestingly enough, the metabolism of one of our patients with pituitary myxedema was elevated fifteen to twenty points by therapeutic procedures aimed only at raising the levels of the blood sodium and chloride.

In myxedema the excretion of 17-ketosteroids in the urine—important evidence of adrenocortical function—is low (1 to 2 mg. in twenty-four hours). In pituitary myxedema the urine is practically free of ketosteroids. In the one the low excretion is probably due to the nonspecific effect of retarded metabolism of adrenal cells; in the other the absence of excretion is due to specific depression of adrenocortical function.

Experimental results from different sources all agree that administration of thyroid causes increased sensitivity of the organism to epinephrine. In the human subject this fact is the basis of the Goetsch

126. Aron, Max; Van Caulaert, C., and Stahl, J.: L'équilibre entre l'hormone préhypophysaire et l'hormone thyroidienne dans le milieu intérieur à l'état normal et à l'état pathologique, Compt. rend. Soc. de biol. **107:** 64 (May) 1931.

127. Kuschinsky, G.: Ueber die Bedingungen der Sekretion des thyreotropen Hormons der Hypophyse, Arch. f. exper. Path. u. Pharmakol. **170:** 510, 1933.

128. Uotila, U. U.: The Regulation of Thyrotropic Function by Thyroxin After Pituitary Stalk Section, Endocrinology **26:** 129 (Jan.) 1940.

129. Belasco, I. J.: Personal communication to the author.

130. Bulger, J. A., and Barr, D. P.: Metabolic Studies of Pituitary Insufficiency, Endocrinology **20:** 137 (March) 1936.

131. Mahoney, William, and Sheehan, Donald: The Effect of Total Thyroidectomy upon Experimental Diabetes Insipidus in Dogs, Am. J. Physiol. **112:** 250 (June) 1935.

132. Swann, H. G., and Johnson, P. E.: Thyroid Function in Diabetes Insipidus in the Rat, Endocrinology **24:** 397 (March) 1939.

133. Marine, David, and Baumann, E. J.: Influence of Glands with Internal Secretions on Respiratory Exchange: Further Data on Effect of Suprarenal Insufficiency (by Removal) in Rabbits, J. Metabolic Research **2:** 1 (July) 1922.

134. Davis, J. E., and Hastings, A. B.: Relationship of Adrenal and Thyroid Glands to Excised Muscle Metabolism, Am. J. Physiol. **105:** 110 (July) 1933.

135. Ingle, D. J., and Higgins, G. M.: The Effect of Thyroxine on the Extent of Regeneration in the Enucleated Adrenal Gland of the Rat, Endocrinology **23:** 419 (Oct.) 1938.

136. Hoen, A.; Langefeld, H., and Oehme, C.: Über die Beziehungen zwischen Schilddrüse und Nebennieren, Endokrinologie **21:** 305, 1939.

137. Bock, K. A.: Die Einwirkung von Nebennierenrindenextrakt auf den Ablauf der Thyroxinmetamorphose bei Froschlarven und beim Axolotl, Klin. Wchnschr. **17:** 1311 (Sept.) 1938.

138. Evans, H. M.; Luck, J. M.; Pencharz, R. I., and Stoner, H. C.: The Calorigenic Action of Amino Acids in the Hypophysectomized Animal, Am. J. Physiol. **122:** 533 (May) 1938.

test for hyperthyroidism. At one time the author measured the response of the metabolism, pulse rate and blood pressure of patients with hyperthyroidism and with myxedema to a standard amount of epinephrine given intravenously. The changes were much greater in the group with hyperthyroidism than in the myxedematous group.[139] This exaggerated sensitivity of patients with hyperthyroidism to epinephrine, so suggestive of underfunction of the adrenal medulla, is at variance with the statement of Crile [140] that overfunction of the adrenal medulla is important in the genesis of exophthalmic goiter.

Gonads.—The evidence of a truly specific interrelationship between the thyroid and the gonads is conflicting. In hyperthyroidism the libido may be increased; in myxedema, diminished. This is not necessarily an expression of endocrine imbalance, but rather of the level of nervous irritability. In myxedema the gonadal function is present but low. This is indicated by the low urinary excretion of 17-ketosteroids (partly of gonadal origin in the male) and of estrogen. Administration of thyroid increases the output of these substances. In cretinism retardation of genital development is the rule. Similarly, thyroidectomy in young rabbits leads to sexual retardation.

Either hyperthyroidism or hypothyroidism may decrease fertility and produce abortion. The striking benefit obtained by us in the treatment of habitual abortion with thyroid is discussed by Means.[1] Similarly King and Herring [141] reported benefit from the treatment of habitual abortion with thyroid.

The characteristic menstrual pattern in thyrotoxicosis is oligomenorrhea or amenorrhea, and in myxedema before the menopause, menorrhagia. These symptoms may be explained on the basis of the nonspecific action of thyroid hormone on gonadal cells, or, in the light of recent experiments, on the basis of a specific effect on the gonads. Several investigators have shown that thyroxin causes degeneration of the ovaries. Uotila [128] found that thyroxin produces atrophy of the seminal vesicles of the rat, probably by depressing the output of gonadotropins by the anterior lobe of the pituitary. Smelser [142] explained such findings by an increase in the threshold of response to gonadotropic and androgenic hormones in animals with hyperthyroidism. Tyndale and Levin [143] concluded that thyroid hormone exerts an inhibiting action on the gonads directly. In any case the action of thyroxin may be such as to produce a state of low estrogenic function. In myxedema the reverse of this situation will be true—a state of relative "hyperestrinism" (metropathia hemorrhagica) is produced.

Recently we have observed several patients with myxedema due to primary dysfunction of the thyroid who had amenorrhea not attributable to the menopause. The hormonic balance, as far as the usual tests show, was not unusual. On administration of thyroid the ovarian cycles of these patients became normal. The most likely explanation is that the ovarian cycle in such cases is normal but the flux of hormones is at a low level (as a result of the low metabolism of the cells). Consequently the phases of endometrial congestion and proliferation are insufficient to produce clinical menstrual bleeding. Thyroid, by stimulating the ovary, increases the production of estrogen and progestin, and the normal cycle returns. This conception fits with the observations of Grumbrecht [144] that thyroid increases the weight of ovaries of infantile rats receiving a constant dose of gonadotropic substance, the increase in weight being proportional to the dose of thyroid.

The evidence of gonadal influence on the thyroid is less conclusive. Gessler [145] showed that estrogen lowers the basal metabolic rate of normal guinea pigs and hypophysectomized rats, and Sherwood [146] found that estrogen reduces the duration of thyroid intoxication of rats. According to Smelser,[142] the amount of androgen necessary to stimulate accessory sex organs of castrates is increased fivefold in thyroxin-treated animals. In fact, Starr and Patton [147] induced remissions in exophthalmic goiter by injecting an extract of pregnancy urine. They suggested that the excess of estrogen produced by the gonadotropic material from pregnancy urine depressed the thyroid directly. On the other hand, Marine [72] stated that "total removal of the gonads in the dog, rabbit and rat usually leads to a slow involution of the thyroid in about one month and to slight reduction of total metabolism." This is confirmed by the results of Ross.[148] Moreover, it has been noted by several workers that estrogen causes a rise in metabolism in normal and in ovariectomized animals; androgen also is said to stimulate the thyroid (Nathanson and collaborators [149]). These discrepancies may be explained by the observation of Pincus and Werthessen [150] that injections of estrogen into animals over a short time (five to ten days) lead to thyroid enlargement whereas injections over a longer time (twenty days or more) lead to thyroid involution. Such observations plus the finding of atrophy of the interstitial cells in a patient with myxedema [151] led Marine to suggest that gonadal atrophy may play a role in the etiology of myxedema. In our own experience castration does not lead to the development of thyroid disease.

Pancreas.—The interrelationship of the pancreas and the thyroid has already been touched on. The well known deleterious effect of thyroid on the severity of diabetes mellitus may not be due to a specific antagonism between thyroid and insulin but may be the result of the characteristic action of thyroid in increasing metabolic rate. Some observers believe that the thyroid and pancreas are antagonistic. Bodansky [152] reported

139. Lerman, Jacob: Unpublished data.

140. Crile, George: The Comparative Anatomy of the Thyroid-Adrenal-Sympathetic System, with Discussion of Unique Function of Adrenal-Sympathetic System, Tr. Am. A. Study Goiter, 1934, p. 23.

141. King, E. L., and Herring, J. S.: Hypothyroidism in the Causation of Abortion, Especially of the "Missed" Variety, J. A. M. A. **113:** 1300 (Sept. 30) 1939.

142. Smelser, G. K.: Testicular Function and the Action of Gonadotropic and Male Hormones in Hyperthyroid Male Rats, Anat. Rec. **73:** 273 (March) 1939.

143. Tyndale, H. H., and Levin, Louis: Ovarian Weight Responses to Menopause Urine Injections in Normal, Hypophysectomized and Hypophysectomized Thyroxin-Treated Immature Rats, Am. J. Physiol. **120:** 486 (Nov.) 1937.

144. Grumbrecht, P.: Die Anwendung von Schilddrüsenpräparaten bei Periodenstörungen, Zentralbl. f. Gynäk. **63:** 1942 (Sept. 2) 1939.

145. Gessler, C.: Influence of Folliculin on the Basal Metabolic Rate, Arch. internat. de pharmacodyn. et de therap. **54:** 263 (Oct. 31) 1936.

146. Sherwood, T. C.: The Relation of Estrogenic Substance to Thyroid Function and Respiratory Metabolism, Am. J. Physiol. **124:** 114 (Oct.) 1938.

147. Starr, Paul, and Patton, Helen: The Effect of Pregnancy Urine Extract and Ovarian Follicular Hormone on Hyperthyroidism, Endocrinology **18:** 113 (Jan.-Feb.) 1934.

148. Ross, R.: Die Beziehungen der Schilddrüse zur Fortpflanzung, Arch. f. Entwcklngsmechn. d. Organ. **137:** 773, 1938.

149. Nathanson, I. T.; Brues, A. M., and Rawson, R. W.: Effect of Testosterone Propionate upon Thyroid and Parathyroid Glands of Intact Immature Female Rats, Proc. Soc. Exper. Biol. & Med. **43:** 737 (April) 1940.

150. Pincus, Gregory, and Werthessen, N.: Continued Injection of Oestrin into Young Rats, Am. J. Physiol. **103:** 631 (March) 1933.

151. Marine, David: Changes in the Interstitial Cells of the Testes in Gull's Disease, Arch. Path. **28:** 65 (July) 1939.

152. Bodansky, A.: Effect of Thyroidectomy upon the Reaction of Sheep to Insulin, Proc. Soc. Exper. Biol. & Med. **21:** 46 (Oct.) 1923.

that thyroidectomized sheep are more sensitive to insulin than normal animals. Conversely, thyroid administered to thyroidectomized rabbits decreases the hypoglycemic action of insulin. He attributed these results to the action of thyroid in promoting glycogenolysis. In our own experience the action of insulin on the blood sugar in myxedema is as follows: The drop in blood sugar from the fasting level requires forty-five to sixty minutes, against a normal of twenty to thirty minutes; the level of depression may be as low as in the normal or lower; the return of the blood sugar to fasting levels requires two hours or more as against a normal of ninety minutes. These results may easily be explained by the fact that all metabolic processes (including the burning of sugar, storage of glycogen and mobilization of epinephrine) are slow in hypothyroidism.

Parathyroid.—There is little evidence of interrelationship between the thyroid and the parathyroid glands. The effect of thyroid on the calcium and phosphorus metabolism is undoubtedly due to the rate of turnover of these elements between bones, blood and kidneys. Consequently, thyroid administration (Aub and associates [153]) or spontaneous thyrotoxicosis (Cope and Donaldson [154]) may raise the level of blood calcium in hypoparathyroidism.

Thymus.—It is well known that the endocrinology of the thymus is yet to be discovered. A relationship between the thyroid and the thymus is suggested by the work of Gudernatsch,[73] who found that tadpoles fed thymus show acceleration of growth and failure of metamorphosis whereas those fed thyroid show the reverse. Speidel [155] noted that feeding thyroid to tadpoles causes proliferation of the thymus. The enlarged thymus in the patient with exophthalmic goiter is a well known finding. Similarly thyroidectomy hastens involution of the thymus (Marine and co-workers; [156] Chiodi [157]). On the other hand, Richter and Wislocki [158] reported evidence contradicting the aforementioned observations. In hypophysectomized rats, whereas the thyroid and adrenals are hypoplastic, the thymus and lymph nodes are hyperplastic.

THYROID AND NUTRITION

It has long been known that thyroid hypertrophy and hyperplasia develop in animals on a high fat or a high protein diet. Rabbits kept on a cabbage diet (Chesney, Clawson and Webster [159]) or a diet of alfalfa hay and oats (Marine and Baumann [160]) and rats kept on a soybean diet (Sharpless, Pearsons and Prato [161]) develop thyroid enlargement. Similarly, animals deficient in iodine or in vitamin D have hyperplastic glands. Conversely, the thyroids of undernourished animals undergo involution—the cells become flat and the follicles distended with colloid (Rabinovitch; [162] Stephens [163]). Stephens offered the interesting suggestion that the changes in inanition are due to suppression of thyrotropic hormone.

In general, the metabolism of vitamins, as well as the metabolism of ordinary food substances, is regulated by the thyroid. Consequently, the need for vitamins parallels the rate of metabolism. Himwich and co-workers [164] showed that vitamin B spares the weight loss induced by thyroid. In patients with hyperthyroidism vitamin B in the form of yeast does not cause reduction in metabolism but improves the appetite and causes weight gain.[165] The author recently observed in a patient, a lifelong "imbiber," that acute peripheral neuritis developed as soon as hyperthyroidism developed. According to Drill,[166] there is no lack of vitamin B_1 in hyperthyroid animals nor is the urinary excretion of this vitamin affected. Such results are to be explained by the increased intake of food by such animals.

There is a marked reduction in the vitamin C content of tissues of rats made toxic with thyroid (Sure and Theis [167]), and excretion of vitamin C is diminished in patients with hyperthyroidism (Lewis [168]).

Several investigators have reported an antagonism between vitamin A and thyroxin. The literature on this subject was reviewed by Wohl and Feldman,[169] who reported pathologic dark adaptation in hyperthyroidism; they attributed this to rapid destruction of vitamin A. They also reported pathologic dark adaptation in myxedema, which they attributed to failure of conversion of carotene to vitamin A in the absence of thyroid hormone. Similarly, carotene diminishes the metabolic effect of thyroid (Smith and Perman [170]). On the other hand, Sure and associates [171] reported marked loss of weight of the thyroid and of other endocrine glands in vitamin A deficiency and in riboflavin deficiency, and Cutting and Robson [172] found that none of the vitamins (A, B_1, B_2, C and D) have any effect on the metabolic rate of guinea pigs with experimental hyperthyroidism induced by a thyrotropic substance.

153. Aub, J. C.; Albright, Fuller; Bauer, Walter, and Rossmeisl, Elsie: Studies of Calcium and Phosphorus Metabolism: VI. In Hypoparathyroidism and Chronic Steatorrhea with Tetany with Special Consideration of the Therapeutic Effect of Thyroid, J. Clin. Investigation **11:** 211 (Jan.) 1932.

154. Cope, Oliver, and Donaldson, G. A.: Relation of Thyroid and Parathyroid Glands to Calcium and Phosphorus Metabolism: Study of a Case with Coexistent Hypoparathyroidism and Hyperthyroidism, J. Clin. Investigation **16:** 329 (May) 1937.

155. Speidel, C. C.: Studies of Hyperthyroidism: II. The Significance of Changes in the Thymus Glands of Thyroid-Treated Frog Tadpoles, Am. J. Anat. **37:** 141 (March) 1926.

156. Marine, David; Manley, O. T., and Baumann, E. J.: Influence of Thyroidectomy, Gonadectomy, Suprarenalectomy, and Splenectomy on Thymus Gland of Rabbits, J. Exper. Med. **40:** 429 (Oct.) 1924.

157. Chiodi, H.: Acción de la tiroidectomia y suprarrenalectomia sobre el timo de ratas albinas castradas, Rev. Soc. argent. de biol. (no. 4) **14:** 322 (July) 1938.

158. Richter, C. P., and Wislocki, G. B.: Anatomical and Behavior Changes Produced in the Rat by Complete and Partial Extirpation of the Pituitary Gland, Am. J. Physiol. **95:** 481 (Nov.) 1930.

159. Chesney, A. M.; Clawson, T. A., and Webster, B.: Endemic Goitre in Rabbits; Incidence and Characteristics, Bull. Johns Hopkins Hosp. **43:** 261 (Nov.) 1928.

160. Marine, David, and Baumann, E. J.: Further Studies on the Etiology of Goiter: The Effect of Cyanides, Tr. A. Am. Physicians **47:** 261, 1932.

161. Sharpless, G. R.; Pearsons, Janice, and Prato, Geneva S.: Production of Goiter in Rats with Raw and with Treated Soy Bean Flour, J. Nutrition **17:** 545 (June) 1939.

162. Rabinovitch, J.: Effect of Underfeeding on Proliferative Activity of Thyroid Gland in Guinea Pig, Am. J. Path. **5:** 87 (Jan.) 1929.

163. Stephens, D. J.: The Effect of the Thyrotropic Principle of the Anterior Pituitary on the Thyroid of the Undernourished Guinea Pig, Endocrinology **26:** 485 (March) 1940.

164. Himwich, H. E.; Goldfarb, Walter, and Cowgill, G. R.: Studies in the Physiology of Vitamins: XVII. The Effect of Thyroid Administration upon the Anorexia Characteristic of Lack of Undifferentiated Vitamin B, Am. J. Physiol. **99:** 689 (Feb.) 1932.

165. Lerman, Jacob, and Hertz, Saul, cited by Means.[1]

166. Drill, V. A.: The Effect of Experimental Hyperthyroidism on the Vitamin B_1 Content of Some Tissues, Am. J. Physiol. **122:** 486 (May) 1938.

167. Sure, Barnett, and Theis, R. M.: Influence of Hyperthyroidism on Vitamin C Content of Various Endocrines and Tissues, Endocrinology **24:** 672 (May) 1939.

168. Lewis, R. A.: The Effect of Hyperthyroidism upon the Metabolism of Vitamin C, Bull. Johns Hopkins Hosp. **63:** 31 (July) 1938.

169. Wohl, M. G., and Feldman, J. B.: Vitamin A Deficiency in Disease of the Thyroid Gland: Its Detection by Dark Adaptation, Endocrinology **24:** 389 (March) 1939.

170. Smith, D. C., and Perman, J. M.: Effect of Thyroxin Combined with Carotene upon the O_2 Consumption of Cats, Endocrinology **27:** 110 (July) 1940.

171. Sure, Barnett; Theis, R. M., and Harrelson, R. T.: Vitamin Interrelationships: I. Influence of Avitaminosis on Ascorbic Acid Content of Various Tissues and Endocrines, J. Biol. Chem. **128:** 245 (July) 1939.

172. Cutting, W. C., and Robson, G. B.: Effects of Various Agents on the Metabolic Rate in Experimental Hyperthyroidism, J. Pharmacol. & Exper. Therap. **66:** 389 (Aug.) 1939.

Council on Pharmacy and Chemistry and the Council on Physical Therapy

QUESTIONS RELATING TO THE METHOD FOR ADMINISTERING ACETYL-BETA-METHYLCHOLINE (MECHOLYL, MERCK & COMPANY, INC.) WHICH AROSE DURING THE COUNCIL ON PHARMACY AND CHEMISTRY'S CONSIDERATION OF THE DRUG WERE REFERRED TO THE COUNCIL ON PHYSICAL THERAPY. THAT COUNCIL VOTED TO GIVE CONSIDERATION TO APPARATUS FOR THE ADMINISTRATION OF DRUGS BY ION TRANSFER AND PREPARED A STATEMENT, WHICH IS REPRODUCED AS THE FIRST PART OF THE FOLLOWING REPORT. THE COUNCIL ON PHARMACY AND CHEMISTRY VOTED TO RECOGNIZE THE USE OF ACETYL-BETA-METHYLCHOLINE CHLORIDE BY THE METHOD OF ION TRANSFER PROVIDED THE FIRM PLACED ON ITS LABELS AND IN ITS ADVERTISING CAUTION STATEMENTS AS STIPULATED. THIS RECOGNITION DOES NOT CARRY WITH IT ANY IMPLICATION OF ACCEPTANCE FOR THE USE OF ANY OTHER DRUGS BY THIS METHOD WITHOUT INDIVIDUAL CONSIDERATION AND ACCEPTANCE BY THE COUNCIL ON PHARMACY AND CHEMISTRY.

THE FOLLOWING REPORT HAS BEEN AUTHORIZED FOR PUBLICATION AFTER CONSIDERATION BY BOTH COUNCILS.

THEODORE G. KLUMPP, M.D., SECRETARY,
COUNCIL ON PHARMACY AND CHEMISTRY.
HOWARD A. CARTER, SECRETARY, COUNCIL
ON PHYSICAL THERAPY.

ION TRANSFER (IONTOPHORESIS)

Direct or galvanic current, in addition to being used for muscle or nerve stimulation, may be used to deposit on or in the tissues ions of certain salts and charged colloid particles from solution. "Ion transfer"[1] is defined as the introduction of soluble salts into the tissues by means of direct current. The term "electrophoresis" applies to a similar movement of colloid particles which are electrically charged or which have absorbed charged particles. The salts of heavy metals such as copper, zinc or tin are frequently used, the positively charged ions moving through the tissues toward the cathode when the current is appropriately passed. Locally these ions may produce precipitation of protein, causing chemical destruction of the tissues. This form of treatment has been used in lesions of the mucous membrane in the practice of proctology, otorhinology and gynecology. The introduction of certain drugs into the tissues by the method of ion transfer (iontophoresis) may also be used to produce systemic effects.

The galvanic current is uninterrupted and unidirectional, and for therapeutic purposes the usual sources are the galvanic wall plate, the motor generator, the valve tube rectifier or the dry cell battery. The efficacy of ion transfer depends on greater penetration of medicaments into the tissue than is obtained by topical application. The depth and local concentration of the medicaments depend on at least two factors. First, although the solution of the medicament is an excellent medium for conducting the current into the tissues, the ions, once introduced, may either become reduced or oxidized to an inactive form or immediately precipitated as proteinates. Second, because of high velocity the ions may be readily eliminated by the body because they are continually swept into the systemic circulation by rapidly moving blood, so that their concentration is too low to be effective. These factors explain why the penetration of medicaments is limited to a few millimeters at most.

The following technic is suitable for introducing drugs by ion transfer: The active electrode should consist of material such as asbestos cloth, cotton gauze, stockinet or other suitable fabric resistant to tears, saturated with a solution of the desired medicament. The saturated fabric is wrapped closely around the part to be treated, and a strip or sheet of metal foil cut to the appropriate size and shape is wound over the fabric. The positive pole of the current source is connected to one end of the metal strip which is used to conduct the current evenly over the entire area treated. The negative pole is connected to a dispersive electrode applied to the patient's back, which completes the circuit. The dispersive electrode consists of a large, flat, moistened pad measuring about 10 by 12 inches. The direct current should be turned on and increased gradually until the desired milliamperage is reached, so that unpleasant effects caused by sudden increases are avoided. The rate and amount of the ion transfer may be regulated by the strength of the current and the duration of its flow respectively.

Individual tolerance to the treatment should serve as a guide in determining the strength of the current to be used. The initial treatment should not exceed 5 or 10 milliamperes for thirty minutes. At the conclusion of a treatment the current is reduced gradually to zero reading and turned off. The patient should remain quiet and warm for thirty to sixty minutes following a treatment; after this period he may resume his usual activities. Each treatment should be restricted to a limited area, such as one hand or one joint when several parts are involved. Three or four days appears to be the most satisfactory interval between treatments.

In the treatment of varicose ulcers, the foot and leg as high as the knee are wrapped appropriately in fabric saturated with medicament and the technic described is then followed. Under no circumstances should the moistened fabric or the metal plate be applied directly over the unhealed ulcerated region. Treatment is usually given two or three times a week but in some cases may be repeated daily.

PRECAUTIONS

The following precautions in the application of ion transfer should be observed:

1. If the sensation of the patient's skin is not normal on test, the current strength must be proportionally selected and carefully controlled according to the size of the electrodes and the milliampere reading.

2. Electrodes must not be applied over denuded areas and must be very carefully applied over recent scar tissue.

3. Electrodes must be sufficiently large to avoid excessive current density.

4. The metal plate of the electrode must be evenly covered by the padding, leaving no bare edges in contact with the skin.

5. The pad covering the electrode must be evenly saturated with the appropriate solution.

6. The electrodes must be applied in good contact by means of even pressure. Tight bandaging, folds, creases and insufficiently moistened areas in the pads may cause nonuniform distribution of current, subjectively manifested first by a local burning sensation.

7. The conducting wires must be fastened securely to the electrodes, so that the metal of the wire tip does not come in contact with the skin.

8. The current must be increased slowly at the start; while the milliammeter registers flow of current, the switch must never be opened or closed sharply.

9. The patient must be instructed to report at once any excessive burning or pain. The judgment of patients as to sensation of burning or pain varies with the individual and, regardless of the milliammeter reading, no more current than the patient states is comfortable should be applied. A safe milliammeter reading must never be exceeded even if the patient states that he can stand more current. During a treatment, the milliammeter reading usually increases as skin resistance decreases; a decrease usually indicates that the pads have become partially dry. In remoistening of the pads the current must be turned off, after which the electrodes are removed, and the pads are moistened evenly and carefully reapplied.

10. The strength of the current must be decreased if at any time the patient complains of annoying sensations. If this reduced current does not provide relief, the current must be turned off, the electrodes removed and the cause of irritation determined.

ACETYL-BETA-METHYLCHOLINE (MECHOLYL)

A 0.2 to 0.5 per cent solution of acetyl-beta-methylcholine chloride (mecholyl chloride) may be introduced by ion transfer. (The Councils point out that a 1:1,000 (0.1 per cent) solution of histamine acid phosphate introduced by ion transfer will

1. Although "ion transfer" is the preferred term, introduction of drugs by this method for the production of local or systemic effects is sometimes called "iontophoresis," "common ion transfer," "ionization," "medical ionization," "ionic medication" or "therapeutic ionization." It is electrochemically incorrect and clinically misleading to term this method "surgical ionization" when a caustic effect is produced.

produce a nearly identical effect.) The disadvantages in administering mecholyl chloride by ion transfer are (1) difficulty in controlling the dosage, (2) costliness of the drug and (3) administration expense—it requires thirty minutes of an attendant's time.

From a review of the evidence now available, the Council on Pharmacy and Chemistry reached the following conclusions:

1. Local effects of mecholyl chloride on the extremities are best obtained when the drug is administered by the method of ion transfer.

2. General effects of mecholyl chloride may be observed during ion transfer, but these are less pronounced when the drug is given by this method than when the subcutaneous route is employed; general effects of a serious or dangerous nature have not been observed when the drug has been given by this method.

3. Mecholyl chloride may be used by this method without obvious harm and with considerable benefit in some cases of chronic ulcers, Raynaud's disease, scleroderma and certain vasospastic conditions of the extremities.

4. The drug used by this method appears to have a palliative effect on the discomfort in some cases of chronic rheumatoid arthritis.

The Council on Pharmacy and Chemistry decided that ion transfer as a method of administering drugs be recognized by the Council with the understanding that the application of the method must at present be reserved to those especially trained and further that the recognition of the method as such carries with it no implication of acceptance of its use for any particular drug.

Council on Pharmacy and Chemistry

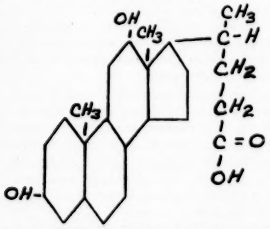
PRELIMINARY REPORT OF THE COUNCIL

THE COUNCIL HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORTS.

THEODORE G. KLUMPP, M.D., Secretary.

DEGALOL (DEOXYCHOLIC ACID)

Under the trade name Degalol, Riedel-de Haen, Inc., submitted for consideration of the Council a preparation of pure deoxycholic acid derived from ox bile, proposed for use in aiding fat digestion and absorption of fat and the fat soluble vitamins A, D and K. According to information furnished by the firm, the product is marketed in 1½ grain tablets flavored with oil of peppermint and is recommended in doses of two tablets three times daily. Deoxycholic acid has the following structural formula:



The firm's submitted advertising includes a bibliography of references to published papers in support of claims that, in addition to a mild cholagogue and choleretic influence, Degalol (deoxycholic acid) is the most effective bile acid for promoting the intestinal absorption of fats. It is claimed that "The isolation of deoxycholic acid and the elimination of ineffective bile acids makes it possible to give smaller and more effective doses of Degalol than of 'bile salts' derived either from ox bile or hog bile" and that "This is of particular importance in a jaundiced patient where the administration of large doses of 'bile salts' increases intrahepatic bile pressure and raises still further the contents of bile acids, and possibly bile pigments, in the blood."

A review of the available evidence concerning deoxycholic acid reveals that the terms "choleic acid" and "deoxycholic acid" were used indiscriminately for three decades until Wieland and Sorge [1] discovered that choleic acid was a coordination compound of eight molecules of deoxycholic acid with one molecule of a fatty acid and found that the smaller molecular component is held so tenaciously that it can only partly be removed by heating in a high vacuum for several days. This concept was introduced by these observers as the "choleic acid principle," which not only eliminated the previous incongruities concerning these bile acids but confirmed the earlier observations of other investigators who had noted the tenacity with which fatty acids are retained whenever purification of bile acids is attempted.

Sobotka [2] pointed out that the significance of this principle for the theory of intestinal resorption is obvious and that this peculiarity of deoxycholic acid to form coordination compounds is shared only to a limited extent by other bile acids. Boedecker and Volk [3] discovered, however, that alpha and beta apocholic acids form coordination compounds very similar to the choleic acids with deoxycholic acid. This additional evidence that the ability of deoxycholic acid to form coordination compounds is shared to an equal extent by apocholic acid is more recently recognized by Sobotka.[4] This author states that it should be kept in mind that animal bile contains conjugated bile acids and that no choleic acids with conjugated bile acids have been isolated. He points out, however, that the conjugated bile acids as found in animal bile share the ability of the free acids of keeping water-insoluble substances in solution and, owing to their greater acidity, retain this dissolving power even in slightly acid reaction down to p_H 6. The latter is significant when it is realized that the contents of the proximal portion of the intestinal tract have a slightly acid reaction.

Wieland and Revery [5] have shown that deoxycholic acid is present in the bile of man and Sobotka,[6] that it is present to greater or lesser extent in the bile of other animals. The latter has also indicated that absorption of ingested cholesterol and of cholesterol excreted by the intestinal mucosa requires the presence of bile.

Numerous studies have been made to show the effect of various bile acids on the solubility and absorption of cholesterol. Schoenheimer,[7] by analysis of intestinal residue in mice and by appearance of lipemia in rabbits, found that the absorption of cholesterol and fats was hastened by the ingestion of deoxycholic acid. Diffusion experiments by Schoenheimer and Hrdina [8] and by Spanner and Bauman [9] demonstrate a stronger association of cholesterol with deoxycholic acid than with any other bile acid. The latter investigators,[9] by the Windaus procedure, have shown that the solubility of cholesterol is about four times as great in a 3 per cent solution of the sodium salt of deoxycholic acid as in the same strength solution of the sodium salt of cholic, taurocholic or glycocholic acid. Andrews and the former investigators [10] have indicated that the faculty of deoxycholic acid to hold cholesterol in solution seems to be also dependent on its ability to form addition compounds with cholesterol.

Bashour and Bauman [11] have reported additional data to confirm the greater effect of sodium deoxycholate on the solubility of cholesterol as compared to sodium cholate and the conjugated bile salts. They point out that the differences between conjugated and unconjugated salts appear to be purely quanti-

1. Wieland, H., and Sorge, H.: Ztschr. f. physiol. Chem. **97:** 1, 1916.

2. Sobotka, H.: The Chemistry of the Bile Acids and Related Substances, Chem. Rev. **13:** 311 (Dec.) 1934.

3. Boedecker, F., and Volk, H.: Ber. d. Deutschen chem. Gesellsch. **53:** 1852, 1920; **54:** 2302, 1921.

4. Sobotka, Harry: The Chemistry of the Sterids, Baltimore, Williams & Wilkins Company, 1938, p. 128.

5. Wieland, H., and Revery, G.: Untersuchungen über die Gallensauren: XXI. Zur Kenntnis der menschlichen Galle, Ztschr. physiol. Chem. **140:** 186, 1924.

6. Sobotka, H.: Physiological Chemistry of Bile, Williams and Wilkins Co., Baltimore, 1937, pp. 119, 125.

7. Schoenheimer, R.: Increasing Cholesterol Absorption with Deoxycholic Acid, Bio. Ztschr. **147:** 238, 1924.

8. Schoenheimer, R., and Hrdina, L.: Etiology of Gallstones: I. Chemical Factors, Proc. Soc. Exper. Biol. & Med. **28:** 944, 1931.

9. Spanner, G. O., and Bauman, L.: The Behavior of Cholesterol and Other Bile Constituents in Solutions of Bile Salts, J. Biol. Chem. **98:** 181, 1932.

10. Andrews, E.; Schoenheimer, R., and Hrdina, L.: Chemical Factors and the Role of the Gallbladder: Etiology of Gallstones, Arch. Surg. **25:** 796 (Oct.) 1932.

11. Bashour, J. T., and Bauman, L.: The Solubility of Cholesterol in Bile Salt Solutions, J. Biol. Chem. **121:** 1 (Oct.) 1937.

tative, the latter being better solvents. Colp and Doubilet[12] have demonstrated the presence of large amounts of deoxycholic acid in human bile but Doubilet[13] has shown that in man the administration of cholic acid is more effective than the administration of deoxycholic acid in raising the concentration of bile acids in hepatic bile. Most of the studies of the effects of bile acids on the solubility of cholesterol have been made in connection with the theory of gallstone formation and are therefore of significance from the standpoint of intestinal absorption only to the extent that cholesterol is similar to fat-soluble vitamins and fats.

The effect of various bile acids on the absorption of fats from the intestinal tract was previously studied by Pohl.[14] The comparative figures obtained by this investigator for the absorption of fat from the intestines of rabbits are 1.22 for sodium cholate and 1.7 for deoxycholic acid, indicating that the latter was 39 per cent more effective than sodium cholate.

Greaves and Schmidt[15] have reported evidence to indicate that bile salts are necessary for the intestinal absorption of B-carotene (precursor of vitamin A) and vitamins D and E. This evidence indicates that deoxycholic acid, not taurocholic, glycocholic or dehydrocholic acid, is responsible for the absorption of B-carotene. Ivy and Berman[16] point out that such clearcut information is not available in the case of vitamins D, E and K. These investigators state that a sufficient amount of comparative evidence does not exist at present for them to state which preparations of bile or bile salts are better for oral therapy. They indicate that while the argument that the salts of oxidized bile acids are preferable to the unoxidized bile acids for oral therapy because the oxidized bile acids show less systemic toxicity may be partly true, this has not been established by in vivo experimentation. Dehydrocholic acid is said not to facilitate the action of lipase like most other bile acids.

Crandall and Ivy[17] have recently reported that a marked rise in blood fat occurs in dogs with "internal" biliary fistulas two to five hours after a meal of olive oil when given with 3 Gm. of commercial bile salts, deoxycholic acid or taurocholic acid. No rise occurs without bile salts or with dehydrocholic, cholic and glycocholic acids. Irvin, Kopla and Johnston[18] have reported the results of photoelectric colorimetric determinations of the provitamin, carotene, in the contents of isolated intestinal loops of dogs before and after absorption periods. They find that a concentrated solution of carotene in cottonseed oil is not absorbed without bile and pancreatic lipase, but when given with hog or ox bile, significant amounts are absorbed, and these are greater when pancreatic lipase is added. Sodium deoxycholate is indicated to be the most effective bile salt for this purpose, but the cholate and glycocholate are also found to be effective.

Rhoads[19] finds that deoxycholic acid is present in ox bile salts, usually from 10 to 20 per cent. The latter reports 14 cases of jaundice with prothrombin deficiency in which desiccated ox bile, iron ox bile salts, dehydrocholic acid and sodium deoxycholate were effective in appropriate dosage with vitamin K; sodium deoxycholate probably the most effective per unit of weight. Similar bile salt preparations were found to be effective with vitamin K in most cases of jaundice by Butt, Snell and Osterberg.[20] They indicate that, in patients with primary hepatic damage, prothrombin deficiency occasionally fails to respond to large doses of vitamin K administered orally with bile salts or given intramuscularly. These authors did not note comparative effectiveness in the bile preparations used.

The successful use of bile salts with oral vitamin K in the treatment of prothrombin deficient hemorrhagic states in which there is lack of intestinal bile is also reported by Warner, Brinkhaus and Smith[21] and by Pohle and Stewart[22]; Ravdin[23] obtained similar results by the use of a crude preparation of vitamin K and sodium deoxycholate or lyophile bile. In a recent report of the Council on Pharmacy and Chemistry[24] it is pointed out that the response of prothrombin deficient patients to intramuscular administration of vitamin K is distinctly less rapid than that which follows oral administration and that the former method has no apparent advantage over the latter except when the jaundiced individual may be unable to take or absorb the vitamin by mouth. The usefulness of bile salts with vitamin K in prothrombin deficiency states when failure to absorb the vitamin is due to lack of intestinal bile appears to be adequately supported by published evidence.

SUMMARY

Consideration of the available published evidence indicates that bile salts are apparently essential for the intestinal absorption of fat-soluble vitamins as well as food fats. Evidence that any one particular bile acid or its salt is essential for the intestinal absorption of all or certain fat-soluble substances is less convincing, except possibly in the case of deoxycholic acid for B-carotene, which needs further confirmation. While there is some chemical and experimental evidence to suggest that deoxycholic acid may be the most efficient bile acid in the transport of fatty compounds across the intestinal mucosa further controlled animal and human studies to furnish corroborative in vivo evidence of this possibility are necessary. Existing clinical evidence does not establish the relative efficiency of the various bile acids with respect to the intestinal absorption of fats or fat-soluble compounds. There is evidence to indicate that deoxycholic acid is one of the most toxic of the bile acids, although the toxicity of bile acids when given orally is of much less importance than when given parenterally. Clinical evidence thus far available does not indicate that orally administered deoxycholic acid produces untoward effects even in amounts somewhat larger than the recommended dosage. It is believed that the use of officially recognized bile salt preparations is more rational for the purpose of promoting the absorption of fats and the fat-soluble vitamins at the present time. In the meantime, the development of further studies to indicate what difference in absorptive activity, if any, is possessed by the various bile acids is awaited with interest. The use of chemically pure bile acid or salt compounds as opposed to impure preparations or mixtures is desirable when there is evidence to indicate that the former function equally well.

The Council recognized the term "Degalol" as the firm's proprietary name for its brand of deoxycholic acid on the basis that the firm was the first to make the product commercially available in this country. The Council voted, however, that consideration of Degalol (Riedel de-Haen, Inc.) and the dosage form, Degalol Tablets, 1½ grains, be postponed because the evidence for its value is not sufficient to warrant acceptance of the product at this time.

12. Colp, R., and Doubilet, H.: Differential Analysis of Bile Acids in Human Gallbladder Bile, Arch. Surg. **33:** 913 (Dec.) 1936; Doubilet, H., and Colp, R.: Differential Analysis of Bile Acids in Human Bile from Fistulas, ibid. **34:** 149 (Jan.) 1937.

13. Doubilet, H.: Hepatic Excretion in Man of the Various Bile Acids Following Their Oral Administration, Proc. Soc. Exper. Biol. & Med. **38:** 50 (Feb.) 1937.

14. Pohl, J.: The Physiologic Action of Bile Acids, Ztschr. f. d. ges. exper. Med. **30:** 423, 1922.

15. Greaves, J. D., and Schmidt, C. L. A.: Role Played by Bile in the Absorption of Vitamin D in the Rat, J. Biol. Chem. **102:** 101, 1933; On the Absorption and Utilization of Carotene and Vitamin A in Cholecystocholonostomized Vitamin A Deficient Rats, Am. J. Physiol. **111:** 492 (April) 1935; Relation of Certain Bile Acids to Absorption of B-Carotene in the Rat, Proc. Soc. Exper. Biol. & Med. **36:** 434 (May) 1937; Relation of Bile to Absorption of Vitamin E in the Rat, ibid. **37:** 40 (July) 1937.

16. Ivy, A. C., and Berman, A. L.: The Rationale of Bile Salt Therapy in Biliary Tract Disease, Minnesota Med. **22:** 815 (Dec.) 1939.

17. Crandall, L. A., and Ivy, H. B.: Bile Salts and Fat Absorption, Abstr.: Rev. Gastroenterol. **7:** 300 (May-June) 1940.

18. Irvin, J. L.; Kopla, J., and Johnston, C. G.: Am. J. Physiol. **132:** 202 (Feb.) 1941.

19. Rhoads, J. E.: The Relation of Vitamin K to the Hemorrhagic Tendency in Obstructive Jaundice, with a Report on Cerophyl as a Source of Vitamin K, Surgery **5:** 794 (May) 1939.

20. Butt, H. R.; Snell, A. M., and Osterberg, A. E.: Further Observations on the Use of Vitamin K in the Prevention and Control of the Hemorrhagic Diathesis in Cases of Jaundice, Proc. Staff Meet., Mayo Clin. **13:** 753 (Nov. 30) 1938; The Preoperative Administration of Vitamin K to Patients Having Jaundice, J. A. M. A. **113:** 383 (July 29) 1939.

21. Warner, E. D.; Brinkhaus, K. M., and Smith, H. P.: Bleeding Tendency of Obstructive Jaundice: Prothrombin Deficiency and Dietary Factors, Proc. Soc. Exper. Biol. & Med. **37:** 628 (Jan.) 1938. Brinkhaus, K. M.; Smith, H. P., and Warner, E. D.: Prothrombin Deficiency and the Bleeding Tendency in Obstructive Jaundice and in Biliary Fistula: Effect of Feeding Bile and Alfalfa (Vitamin K), Am. J. M. Sc. **196:** 50 (July) 1938.

22. Pohle, F. J., and Stewart, J. K.: Observations on Plasma Prothrombin and Effects of Vitamin K in Patients with Liver or Biliary Tract Disease, J. Clin. Investigation **19:** 365 (March) 1940.

23. Ravdin, I. S.: Some Recent Advances in Surgical Therapeusis, Ann. Surg. **109:** 321 (March) 1939.

24. Preliminary Report on Vitamin K: II, J. A. M. A. **113:** 2056 (Dec. 2) 1939.

REPORT OF THE COUNCIL

FOILLE (CARBISULPHOIL COMPANY) NOT ACCEPTABLE FOR N. N. R.

At the request of the Council, the Carbisulphoil Company of Dallas, Texas, submitted under the name "Foille" an emulsion for use as a new protective antiseptic and analgesic dressing in the local treatment of recent and infected burns, traumatic and infected wounds, and indolent ulcers. The product is stated to consist of the following ingredients:

	Approximate Percentage
Alcohol	1.20
Phenol	2.36
Benzocaine	1.26
Calcium soap	0.36
Calcium iodide	0.24
Potassium iodide	0.12
Oxyquinoline base	0.18
Calcium thiosulfate	0.03
Calcium sulfite	0.07
Calcium sulfate	0.13
Sulfur (free)	0.17
Water	2.85
Corn Oil (fixed)	90.33
Glycerin	present

The method of preparation described by the manufacturer involves the use of corn oil, Sulfuretted Calcium Solution, N. F., Phenol, U. S. P. (liquefied by addition of 10 per cent water), Tincture of Iodine, U. S. P., benzocaine and 8-hydroxyquinoline. In proof of the composition of Foille, chemical analyses from two laboratories were submitted. The A. M. A. Chemical Laboratory found the composition to be essentially that claimed.

In reply to numerous inquiries resulting from the wide promotion of Foille as "The New-Type Treatment for Burns," the Council's office has called attention to a somewhat similar product previously considered by the Council, which was submitted in 1929 under the name "Carbisulphoil" by the Globe Laboratories of Fort Worth, Texas. The manufacturer of this product stated that it was supplied in two strengths: "Carbisulphoil Medicine No. 2," prepared by incorporating 2 per cent of 30 per cent phenol, 2 per cent of tincture of iodine, U. S. P., and 2 per cent of sulfurated lime, N. F., with corn oil; and "Carbisulphoil Medicine No. 4," prepared by adding 4 per cent of each of these ingredients to corn oil instead of 2 per cent. It was claimed to be therapeutically indicated for surgical dressings, cuts, wounds, bruises, burns, ulcers, piles and gonorrheal infections in male and female. The Council declared Carbisulphoil unacceptable for N. N. R. because it was an unscientific mixture of indefinite composition marketed with unwarranted claims. Subsequently the Globe Laboratories submitted a chemical analysis of composition of Carbisulphoil No. 2 and No. 4 as follows:

Sample No. 2		*Sample No. 4*	
Potassium iodide	0.1%	Potassium iodide	0.2 %
Calcium iodide	0.3%	Calcium iodide	0.4 %
Calcium thiosulfate	0.1%	Calcium thiosulfate	0.2 %
Calcium tetrathionate	0.2%	Calcium tetrathionate	0.03%
Soap (insoluble)	0.2%	Soap (insoluble)	0.47%
Sulphur (free)	0.3%	Sulphur (free)	0.9 %
Ethyl alcohol	1.5%	Ethyl alcohol	3.0 %
Phenol	1.6%	Phenol	3.2 %
Vegetable oil	94.0%	Vegetable oil	88.0 %
Water	1.7%	Water	3.6 %
Glycerin	trace	Glycerin	trace

After further consideration the Council voted to postpone publication of its report to await further evidence from the firm, provided there was no exploitation of the product beyond giving out experimental supplies to selected physicians. The Globe Laboratories later wrote that because of its inability to make the product acceptable it would neither manufacture nor sell the preparation in the future. The Council, therefore, never published its report, declaring Carbisulphoil unacceptable for New and Nonofficial Remedies.

The Carbisulphoil Company has disclaimed that Foille is essentially the same as its predecessor Carbisulphoil (Globe Laboratories) on the ground that the process of manufacture and therapeutic indications are different for Foille and that Carbisulphoil was an evil-smelling, unstable product and has expressed the view that it is prejudicial to compare the two products. The Council desires to point out, however, that although Foille apparently differs in certain respects from Carbisulphoil it consists, nevertheless, of an even greater number of ingredients, all except two of which are essentially identical with those of its predecessor. From the preceding analyses it will be noted that Foille differs essentially in composition from Carbisulphoil only by the addition of oxyquinoline sulfate and benzocaine. The Council believes it only fair to indicate the points of similarity of Foille and Carbisulphoil in order to show that the former is not an entirely new type of preparation advanced for the treatment of burns.

The manufacturer claims that Foille is not a definite pharmaceutic mixture nor a chemical entity but that its ingredients are suspended in an emulsion and that they are not more numerous than those in other mixtures accepted by the Council. It is promoted as a new and more efficient treatment for burns. The benzocaine plus the phenol is said to effect an immediate and pronounced anesthesia which mitigates shock and pain to provide a distinctly favorable factor in the initial stage of a serious burn. The action of the soap augmented by phenol on prolonged contact is claimed to coagulate the serum exudation, excluding air and forming a soft shield. It is also claimed that the coagulation does not rigidly seal over the burned tissues and permits moderate immediate separation. The oxyquinoline base is stated to provide an efficient bacteriostatic and antiseptic action and to supply stimulation of granulation. The iodide salts are claimed to have a definite therapeutic effect. The claim is made that the potassium iodide is absorbed easily from wound surfaces and that the employment of alkaline iodides is based on empirical foundation. The firm quotes Poulsson, Bastedo, Sollmann and Zwick on the iodides, on which basis the claim is made that iodides may be expected to promote catabolism of the diseased cells and thereby accelerate the healing process. The firm expresses the belief that the iodides in Foille are, to an appreciable degree, partly responsible for the evidence of speedier granulation and epithelization and the pronounced efficiency in treating infected burns and indolent ulcers. However, no convincing evidence has been shown in support of such action by these agents.

In support of its claims for the product the manufacturer submitted F. D. A. Agar Cup Plate tests from three laboratories to indicate the antiseptic action of Foille on Staphylococcus aureus and certain fungi, and additional laboratory statements to show that poisoning from the phenol contained in the product will not occur because it is not appreciably absorbed through the skin because of its affinity for the water-in-corn oil vehicle and that a mild antiseptic action occurs with prolonged application. Bacteriologic examination of the product by a consultant of the Council indicates that Foille exhibits a distinct inhibitory effect on staphylococci and no inhibitory effect on B. coli on plain agar cups, and that it is impossible to say whether such inhibitory effects occur in the case of infected wounds, because a bacteriologic study of them would be difficult to accomplish on account of the nature of the product. It was suggested that clinical tests should be made with controls of Foille in which phenol, oxyquinoline sulfate and the iodides have been omitted. The inhibitory effects of Foille on staphylococci must be accounted for on the basis of its content of phenol and oxyquinoline sulfate, both of which are known to have fair bacteriostatic qualities.

In further support of its claims, the manufacturer submitted 75 case histories and statements (mostly testimonials) from physicians, exhibits of serial photographic human and animal studies, four medical papers, a hospital study compiled by the City-County Hospital, Fort Worth, Texas, three statements by hospital executives, and lists of industrial and other institutions, clinics and hospitals employing Foille. Much of this material appears in a large illustrated advertising brochure entitled "Foille, the New-Type Treatment for Burns" distributed by the manufacturer.

The twenty-two physicians whose case reports appear in the advertising brochure were requested by the Council office to indicate whether or not these reports represented their present views concerning Foille and whether or not they had done any controlled work. In most instances the replies which were received admitted that no controlled work had been done, and such additional information as was given is largely testimonial and does not constitute evidence to establish the claims made for the product. The two series of photographic animal studies submitted partially illustrate the results reported in the paper

by Terrell,[1] reprinted in the firm's advertising brochure. The paper indicates the comparative effects of Foille and 10 per cent tannic acid in the treatment of equally extensive cautery burns in 4 dogs, and toxicity tests of Foille by application to cautery burns and by oral administration in another 4 dogs for a period of twenty-two days. The 2 Foille-treated dogs were stated to be more comfortable and to show more rapid healing than the two tannic acid-treated animals; necropsy of the other four dogs showed only petechiae in the small intestine of the two externally tested animals and no evidence of toxic effects in the two orally tested dogs.

The second medical paper, by Galt,[2] points out that Underhill has rather conclusively proved that toxemia in burns is not due to absorption of products from the burned areas. Apparently this is intended as an argument in favor of the use of Foille as an antiseptic and presumably refers to the previous reports by Underhill and his co-workers,[3] who showed that early toxic symptoms are due to dehydration, increased concentration of the blood and loss of blood chlorides and serum protein. These investigators concluded that an elevation of temperature after the first twenty-four to thirty-six hours is not the result of any absorption of decomposed protein from the burned areas but is caused by infection. This appears to emphasize the need for aseptic precautions and general measures to combat dehydration, rather than a recommendation for the use of a local antiseptic oil such as Foille. Kapsinow[4] has shown experimentally by injection of dyes and lethal doses of strychnine into the burned areas that no absorption from these areas occurs within the first twenty-four to thirty-six hours. While such studies have cast considerable doubt on the toxic theory advanced by Davidson[5] for the use of tannic acid, the results obtained from local use of escharotics in the treatment of burns are still regarded favorably by many investigators. The invalidation of the theory that decomposed protein absorbed from burned areas produces shock simply minimizes the importance of local treatment and does not discredit the principles of systemic treatment to combat burn shock, when this is present, from whatever cause.

A third paper, by Dr. Samuel Webb Jr., read before the Northeast Medical Society on Oct. 10, 1939, simply gives this author's experiences with the use of Foille over a period of eighteen months. Most of the statements made represent personal impressions.

The fourth paper, by Nagel,[6] represents a single case report, which is not adequate to support the author's conclusion that Foille represents an advance in the treatment of major burns. A recently published paper by Noland and Wilson[7] includes photographs of burns to show the results of local treatment with Foille but does not furnish adequate evidence to justify the conclusions made by the authors.

The submitted hospital study reports two groups of burned patients: one group of 20 treated with various types of remedies other than Foille, and another group of 20 treated with the latter. The Foille-treated patients were stated to show marked comfort and reduction in contracture, scarring and infection, and to require comparatively less opiates, sedatives, dressings and hospitalization than patients receiving other types of treatment. From the data given a conclusion is difficult to draw, since burns vary in degree as well as in extent. The submitted statements of hospital executives furnish no actual evidence, and the submitted lists of organizations and physicians employing Foille represent a brief in support of the manufacturer's ability to sell the product rather than proof of its value as a therapeutic agent.

The Council considered an unpublished controlled clinical report made by Dr. Joseph E. Hamilton and Dr. Arnold Griswold on the effects of Foille and tannic acid in the treatment of alternate patients with comparable burns at the Louisville City Hospital, Louisville, Ky. The series of cases comprised 42 patients treated with Foille and 36 treated with tannic acid. These investigators found no evidence of toxicity from the phenolates or other constituents of Foille. They found no essential difference in the control of traumatic shock other than that Foille permitted more initial exudation than did tannic acid and did not seem as efficient as a good tannic acid eschar in relieving pain in the first few days (acute stage) of the burn. No detectable difference in the effect on toxemia was evident. They found that the Foille-treated lesions were less septic than those treated by tannic acid and that, because the soft coagulum produced by Foille may be easily removed with tub baths, its repeated application in the granulating stage of deeper burns was less painful than when tannic acid had been employed. In the more superficial burns, which did not require skin grafting, the tannic acid eschar necessitated less trouble and nursing care in many cases for which no further treatment was necessary. It was also found that the use of Foille in deeper burns permitted earlier skin grafting and apparently had no injurious effect on freshly implanted pinpoint grafts. Scarring and contracture in deep second and third degree burns was somewhat less with Foille; and the hospital stay, although slightly reduced, was not remarkably less than for tannic acid-treated burns.

The Council also gave consideration to reports on the use of other agents employed in the local treatment of burns. Bettman[8] has modified the original tannic acid treatment of Davidson by applying silver nitrate (10 per cent) following an initial application of tannic acid (5 per cent) after all loose skin from opened blebs is removed and believes that this treatment shortens the period for production of an eschar. The eschar produced is unusually firm and impermeable, which is said to prevent edema beneath it, thereby avoiding embarrassment of the circulation and preventing the entrance of infection. Bettman says "As stated earlier, red blood cells and albumin do not occur in the urine of patients under the tannic acid-silver nitrate regimen in the absence of applications of grease or oil; but, when these applications have been made, there is always albumin in the urine, even though an attempt has been made to remove them." He believes that the slight surface moisture produced by oils is enough to permit the entrance into the blood stream of toxins or other elements that are present on the surface and to allow some infection to develop. The factor of dryness seems to be stressed, especially in connection with infection. He states further, "Burns treated with oil and grease heal slowly, become infected, form abundant scar tissue and have a long and trying period of hospitalization."

Blackfield and Goldman[9] are in agreement with the idea of production of a dry eschar in the shortest possible time to prevent infection, and they favor the use of the aseptic tannic acid bath previously described by Wells. They emphatically condemn the use of unguents, which is stated to be a type of initial treatment that may interfere with modern therapy. The tannic acid bath is followed with 10 per cent solution of silver nitrate to produce an immediate tan. The tanned areas, including edges, are then treated with applications of 1 per cent aqueous solution of gentian violet several times daily to keep the eschar dry and prevent infection. They regard their method as the one of choice in the early local treatment of burns. Although they avoid rigid eschar formation because of the danger of constriction of circulation in burns encircling the extremities, they do not recommend the use of oily preparations.

Meyer and Wilkey[10] reported 968 burn cases in the Cook County Hospital, Chicago, treated by different methods. Of

1. Terrell, T. C.: Comparative Study of Foille with Tannic Acid and Tannic Acid Preparations in the Treatment of Burns, Texas State J. Med. **34**: 409 (Oct.) 1938.

2. Galt, Sidney: The Use of Foille in the Treatment of Burns, Dallas M. J. **25**: 81 (July) 1939.

3. Underhill, F. P.; Kapsinow, Robert, and Fisk, M. E.: Studies on Mechanism of Water Exchange in Animal Organism; Nature and Effects of Superficial Burns, Am. J. Physiol. **95**: 302 (Nov.) 1930; Extent of Edema Fluid Formation Induced by Superficial Burn, ibid. **95**: 325 (Nov.) 1930; Relationship of Blood Chlorides to Chlorides of Edema Fluid Produced by Superficial Burn, ibid. **95**: 334 (Nov.) 1930; Composition of Tissues Under Influence of Superficial Burn, ibid. **95**: 339 (Nov.) 1930.

4. Kapsinow, Robert: Rate of Absorption from Extensive Superficial Burns, New Orleans M. & S. J. **85**: 597 (Feb.) 1933.

5. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**: 202 (Aug.) 1925.

6. Nagel, Patrick: Use of Antiseptic Anesthetic Agent Locally in Extensive Burn, Oklahoma State M. J. **33**: 14 (Jan.) 1940.

7. Noland, Lloyd, and Wilson, C. H.: More Recent Ideas in the Treatment of Burns, J. M. A. Alabama **10**: 157 (Nov.) 1940.

8. Bettman, A. C.: The Rationale of Tannic Acid-Silver Nitrate Treatment of Burns, J. A. M. A. **108**: 1490 (May) 1937.

9. Blackfield, H. M., and Goldman, Leon: Burns in Children, J. A. M. A. **112**: 2235 (June 3) 1939.

10. Meyer, K. A., and Wilkey, J. L.: Modern Treatment of Burns, Minnesota Med. **21**: 644 (Sept.) 1938.

272 patients treated with 5 per cent tannic acid aqueous solution, 34 died and 108 showed infection; 104 patients with second and third degree burns were treated with Aldrich's compound aniline dye mixture (by weight: gentian violet 3 parts, brilliant green 2 parts, neutral acriflavine 1 part), with a mortality of 8.7 per cent and infection in only 10 cases; 125 patients with second degree burns were treated with 1 per cent aqueous solution of gentian violet only, with 1.6 per cent mortality and infection in 22 patients; 220 patients with severe second and third degree burns were given Bettman's tannic acid-silver nitrate treatment followed by application of scarlet red to unhealed areas after removal of the crust, with a mortality of 8.6 per cent and infection in only 18 cases; 26 patients with second degree burns of the extremities received the silver nitrate-methyl rosaniline dye local treatment, with no deaths and only 2 cases of infection; 38 patients with inextensive first and second degree burns were treated with crude cod liver oil applications after cleansing, with no deaths; but, with the second degree burns, infection occurred in 8 cases and delayed healing occurred in many instances, and 186 patients with contaminated burns received local saline and boric acid dressings, plus, in many cases, 3 per cent xeroform (tribromphenol plus bismuth) treated sterile petrolatum gauze strips, with only 0.6 per cent mortality (2 deaths). The authors conclude that the Bettman tannic acid-silver nitrate treatment is the local therapy of choice in uncontaminated or noninfected severe burns not more than 12 hours old. They believe the aniline dye treatment is better after infection is imminent; and, most important, the general treatment of initial shock, plus good nursing care, should take precedence over the local therapy.

McClure[11] has recently reported the method employed at Henry Ford Hospital, Detroit, in the management of severe burns. He also presents an excellent review of the accepted methods of treatment, emphasizing the subjugation of local therapy for attention to threatening toxemia, alterations in blood chemistry, a wound very susceptible to infection and pathologic changes in organs remote from the skin. He indicates that the original toxin-absorption theory is not yet settled but points to recent evidence corroborating the early observations that liver necrosis occurs in fatal cases of burns.[12]

The Council's referee desires to emphasize that there are certain physiologic and pathologic processes in connection with burns which require something more than the local application of a "shotgun" soap preparation. Trusler, Egbert and Williams[13] have recently pointed out that the late symptoms of pulmonary edema, emesis, hyperpyrexia, progressive anasarca, convulsions, coma and circulatory collapse which occur after a severe extensive burn, have been falsely ascribed to toxemia and are more probably the result of disturbance in water balance which occurs when fluids are forced and the tissues become waterlogged by fluid loss through the injured capillaries. They emphasize the importance of maintenance of fluid balance and the use of blood and plasma transfusions and moderate amounts of other fluids, with large amounts of dextrose and oxygen, in the treatment of severe burns.

SUMMARY

The evidence submitted by the firm to support the claims for Foille is largely testimonial, poorly, if at all, controlled and consists of considerable reduplication of material. The case reports submitted are further inadequate from the standpoint of blood studies by which to evaluate the systemic condition of the patients. The reported experiments on 4 dogs to determine the toxicity of Foille and mere clinical statements based on negative evidence to the contrary cannot be regarded as adequate. Aside from the question of systemic absorption the possible danger of local injury to tissues by the action of 2.35 per cent phenol should be considered. In view of the bacteriologic laboratory evidence, the firm's claim that the product is antiseptic should be modified; and it is pointed out that this factor cannot be evaluated in the absence of comparative clinical studies using control preparations of Foille in which phenol, oxyquinoline sulfate and the iodides are omitted. The Council believes that such controlled evidence is essential to establish any therapeutic rationale for the large number of ingredients contained in Foille and, by corollary, that the available evidence constitutes no real proof of the firm's claim that the so-called active ingredients all have a therapeutic effect. As has already been pointed out, the pain-controlling effect of Foille is not surprising in view of its phenol and benzocaine content, and the results reported do not appear remarkable or essentially different from those which might be expected with the use of other local agents.

Reports in the literature indicate that the choice of local therapy in the treatment of burns depends on the degree and type of involvement; that the initial use of oily preparations, if not actually harmful, may be detrimental to the use of the more commonly employed escharotic agents in severe burns; and that the treatment of the systemic reactions accompanying severe burns is probably of greatest importance. Available evidence indicates that the use of tannic acid-silver nitrate-antiseptic dye preparations for the production of a firm protective eschar is most widely accepted in the local treatment of severe burns at the present time, irrespective of which of the theoretical factors—absorption of split protein, loss of serum (dehydration) or infection at the site of burn—is mainly responsible for the grave systemic reactions which occur.

The Council does not wish to discourage scientific laboratory and clinical investigation from developing a more efficient local agent for use in the treatment of extensive burns; but in view of the conflicting evidence at the present time it is unwilling to accept a partially antiseptic, complex, oily soap emulsion possessing anesthetic properties for use in the local treatment of burns, wounds and chronic ulcers. The meaningless proprietary name Foille and the inclusion of iodides, or other ingredients, in its formula on an empirical basis are in conflict with the Council's rules. It is believed that more rigidly controlled comparative clinical investigations are necessary to determine not only which, if any, of the ingredients in the product are responsible for its claimed effectiveness in certain cases, but also definitely to establish whether or not the results obtained are essentially different from those secured with other preparations. Not only should cases of approximately equal severity be selected for such studies, but systemic treatment must also be kept as nearly uniform as possible. Control of pain by the administration of opiates or other analgesics in those cases not treated with a local anesthetic preparation should be done to eliminate this factor. Whether or not the addition of anesthetic substance to local agents for burns may obviate the use of opiates appears to depend on evidence to prove the harmlessness and value of viscous oily vehicles, since only in such a medium would the anesthetic be kept in contact with the surface for a sufficient length of time to exert more than transient effects. It is emphasized that the Council's principal objection to Foille, aside from the name, is not primarily that it consists of a large number of ingredients but that there is lack of evidence to establish the therapeutic rationale for a preparation of such complexity. The burden of proof to substantiate the claims made for the product lies with the firm.

The foregoing report (now slightly modified) was transmitted to the manufacturer. It has replied that additional control studies are being set up in several hospitals to obtain the further evidence required by the Council and requested that publication of its report be postponed until more evidence could be obtained. The manufacturer was asked to withdraw active promotion of Foille pending the accumulation of satisfactory evidence of its value, but it has indicated that it is unwilling to do so. Under its procedure, the Council is therefore obliged to publish this statement of its consideration of Foille.

The Council voted that Foille (Carbisulphoil Company) be not accepted for inclusion in New and Nonofficial Remedies because it is marketed under an uninformative proprietary name without satisfactory evidence to establish the therapeutic rationale for its complex formula and the claims made for it.

11. McClure, R. D.: The Treatment of the Patients with Severe Burns, J. A. M. A. **113:** 1808 (Nov. 11) 1939.
12. Belt, T. H.: J. Path. & Bact. **48:** 493 (May) 1939.
13. Trusler, H. M.; Egbert, H. L., and Williams, H. S.: Burn Shock: Water Intoxication as a Complication, J. A. M. A. **113:** 2207 (Dec. 16) 1939.

Council on Foods and Nutrition

THE COUNCIL HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORT.
FRANKLIN C. BING, Secretary.

INGREDIENTS FOR USE BY BAKERS FOR IMPROVING THE NUTRITIONAL VALUE OF BREAD

The Council has encouraged the restorative addition of essential vitamins and minerals to processed foods in order to facilitate the selection of diets that are adequate for optimal health. The nutritional value of bread can be improved by the use either of specially milled flours which contain the major portion of the vitamin and mineral content of the grain with a minimum of coarse bran derived from the outer branny coats or of white flour to which an appropriate mixture of crystalline vitamins (or vitamin concentrates) and mineral salts has been added by the miller. Another satisfactory procedure is the addition by the baker of a suitable vitamin and mineral mixture to the ordinary white flour used in preparing the bread dough. Some considerations involved in the selection of an appropriate vitamin and mineral mixture have been discussed in the Council's report on "Nutritionally Improved or Enriched Flour and Bread." [1]

The following product has been declared accepted by the Council as a convenient preparation for use by bakers for increasing the thiamine, riboflavin, nicotinic acid, iron and calcium contents of ordinary white flour to be used in making "Enriched Bread." Standards for "Enriched Bread" have been suggested and will be discussed at hearings of the Food and Drug Administration to be held in Washington during 1941.

ACCEPTED FOODS

THE FOLLOWING ADDITIONAL FOODS HAVE BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON FOODS OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO ACCEPTED FOODS.
FRANKLIN C. BING, Secretary.

FOODS FOR SPECIAL DIETETIC PURPOSES (*See* Accepted Foods, 1939, page 295).

General Mills, Inc., Minneapolis.

VIBIC BREAD INGREDIENT, a mixture of cornstarch, thiamine hydrochloride, riboflavin (or its concentrate), nicotinic acid, calcium sulfate ($CaSO_4.2H_2O$) and iron phytate. This product is designed for use by commercial bakers as a nutritional ingredient in bread doughs.

Analysis (submitted by manufacturer).—Moisture 12.8%, total solids 87.2%, ash 23.3%, protein (N × 5.7) 4.8%, fat (ether extract) 0.2%, crude fiber 0.3%, carbohydrates other than crude fiber (by difference) 58.6%, calcium (Ca) 5.4%, iron (Fe) 0.66%.

According to reports of thiochrome (Hennessy-Cerecedo method [2]) assay this product contains 310 international units of thiamine per gram, 420 mg. per pound. According to reports of microbiologic (Snell and Strong method [3]) assay this product contains 210 mg. of riboflavin per pound. This product contains 1,230 mg. of nicotinic acid, 3,000 mg. of iron (Fe) and 24,000 mg. of calcium (Ca) per pound. When Vibic bread ingredient is used at the rate of 0.5 per cent in a bread formula (½ pound of Vibic ingredient to 100 pounds of flour) the resulting bread will contain 1.4 mg. of thiamine, 4.0 mg. of nicotinic acid, 0.8 mg. of riboflavin, 11.8 mg. of iron (Fe) and 132 mg. of calcium (Ca) to the pound loaf.

The following product has been declared accepted by the Council as a specially milled flour to be used by bakers in the preparation of "Enriched Bread." Bread made with this flour has a texture similar to white bread, a uniform light brown color and a characteristic flavor. Such bread will meet the standards for thiamine, riboflavin, nicotinic acid and iron contents which have been suggested for "Enriched Bread" and which will be discussed at hearings of the Food and Drug Administration to be held in Washington during 1941.

VIBIC BREAD FLOUR, a mixture of bleached white flour from hard wheat, definite proportions of flours prepared from selected layers of the wheat kernel, riboflavin (or its concentrate), nicotinic acid, calcium sulfate ($CaSO_4.2H_2O$) and iron phytate.

Analysis (submitted by manufacturer).—Moisture from 13.0 to 14.5%, ash (added mineral compounds excluded) not exceeding 1%, fat (ether extract) approximately 2.0%, protein (N × 5.7) from 12.5 to 14.0%, total carbohydrates other than crude fiber (by difference) approximately 70.0%, crude fiber approximately 0.5%, calcium (Ca) 0.045%, iron (Fe) 0.004%.

According to reports of thiochrome (Hennessy and Cerecedo method [2]) assay this product contains not less than 1.75 international units of thiamine per gram, 2.4 mg. per pound. According to reports of microbiologic (Snell and Strong method [3]) assay this product contains not less than 1.2 mg. of riboflavin per pound. This product contains nicotinic acid added in amounts sufficient to produce a total content of nicotinic acid of not less than 6.15 mg. per pound. Bread made with this flour will contain 1.4 mg. of thiamine, 4 mg. of nicotinic acid, 0.8 mg. of riboflavin, 11.8 mg. of iron (Fe) and 132 mg. of calcium (Ca) per pound loaf.

Calories.—3.53 per gram; 100 per ounce.

Orpro Food Products, Los Angeles.

ORPRO WITH ADDED VITAMINS B_1 (THIAMINE) AND G (RIBOFLAVIN), bottled, concentrated apple juice prepared from Rome Beauty, Delicious and Winesap apples; containing added crystalline vitamins B_1 (thiamine) and G (riboflavin).

Analysis (submitted by manufacturer).—Moisture 24.1%, total solids 75.9%, ash 1.5%, fat (ether extract) 0.4%, protein (N × 6.25) 0.4%, crude fiber none, sucrose 15.6%, reducing sugar as invert sugar 48.0%, carbohydrates other than crude fiber (by difference) 71.6%, calcium (Ca) 0.023%, phosphorus (P) 0.065%, iron (Fe) 0.0013%, total acidity as malic acid 1.6%, alcohol precipitate (pectic substances) 0.4%, *pH* 4.0, alkalinity of ash (number of cc. of normal acid required to neutralize ash from 100 Gm. sample) 21.2. Tests of lead and arsenic show that the product complies with Council requirements for freedom from toxic spray residues.

Protocols of biologic assay (1940) indicate that the product contains not less than 250 international units of vitamin B_1 and 375 Sherman-Bourquin units of vitamin G per fluidounce.

Calories.—2.83 per gram; 80 per ounce; 3.76 per cubic centimeter; 111 per fluidounce.

State Mill and Elevator, Grand Forks, N. D.

DAKOTA MAID BRAND WHEAT GERM, mildly heat processed to prevent rancidity.

Analysis (submitted by manufacturer).—Moisture 4.0% to 5.0%, total solids 96.0% to 95.0%, ash 4.2% to 5.2%, protein (N × 6.25) 30.0% to 32.0%, fat (ether extract) 10.0% to 13.0%, crude fiber 1.5% to 2.0%, carbohydrates (other than crude fiber) by difference 50.3% to 42.8%, phosphorus 1.1%, magnesium 0.25%, calcium 0.06%, iron 0.005%.

Reports of biologic assay submitted by the manufacturer (1940) indicate that the product contains not less than 11 international units of vitamin B_1 per gram, 312 per ounce; [1] and not less than 4 Bourquin-Sherman units of vitamin G (riboflavin) per gram; 114 per ounce.[2]

Calories.—4.11 to 4.16 per gram; 116 to 118 per ounce.

GRAIN PRODUCTS (*See* Accepted Foods, 1939, p. 95).

Albers Milling Company, Milwaukee.

ALBERS CARNATION BRAND ROLLED WHEAT ENRICHED WITH VITAMIN B_1, a flaked, cooked wheat with the coarser bran portion removed, with added crystalline thiamine hydrochloride.

Analysis (submitted by manufacturer).—Moisture 11.2%, total solids 88.8%, ash 1.6%, protein (N × 5.83) 9.2%, fat (ether extract) 2.0%, crude fiber 1.5%, reducing sugars as invert sugar 0.1%, sucrose 2.7%, carbohydrates other than crude fiber (by difference) 74.3%, phosphorus (P) 0.31%, iron (Fe) 0.005%, calcium (Ca) 0.037%.

Report of chemical assay (1941) by the thiochrome method, submitted by the manufacturer, indicates a thiamine content of 0.0071 mg. per gram, 0.202 per ounce.

Calories.—3.53 per gram; 100.2 per ounce.

The Concordia Milling Company, Concordia, Kan.

SUNRISE BRAND PANCAKE FLOUR, containing bleached hard wheat flour, mono calcium phosphate ($CaH_4[PO_4]_2.H_2O$), sodium bicarbonate, and sodium chloride.

Analysis (submitted by manufacturer).—Moisture 11.4%, total solids 88.6%, ash 5.8%, fat (ether extract) 1.02%, protein (N × 5.8) 11.8%, crude fiber 0.35%, total carbohydrates other than crude fiber (by difference) 69.6%.

Calories.—3.33 per gram; 95 per ounce.

1. J. A. M. A. **116:** 2849 (June 28) 1941.
2. Hennessy, D. J., and Cerecedo, L. R.: The Determination of Free and Phosphorated Thiamine by a Modified Thiochrome Assay, J. Am. Chem. Soc. **61:** 179 (Jan.) 1939.
3. Snell, G. G., and Strong, F. M.: Riboflavin Assay in Biological Materials by Micro Method, Indust. & Engin. Chem., Anal. Ed. **11:** 346 (June) 1939.

1. Supplee, G. C., and Bender, R. C.: A Standardized Method for the Determination of Vitamin B_1, Indust. & Engin. Chem. Anal. Ed. **10:** 636, 1938.
2. Supplee, G. C.; Bender, R. C., and Jensen, O. G.: Determining Riboflavin—a Fluorometric and Biological Method, Indust. & Engin. Chem. Anal. Ed. **11:** 495, 1939.

INFANT FOODS (*See* Accepted Foods, 1939, p. 156).

Harold H. Clapp, Inc., Rochester, N. Y.

CLAPP'S BRAND STRAINED APRICOTS WITH APPLES.

Analysis (submitted by manufacturer).—Moisture 82.29%, total solids 17.71%, ash 0.41%, ether extract 0.10%, protein (N × 6.25) 0.40%, crude fiber 0.55%, carbohydrate other than crude fiber (by difference) 16.25%.

Calories.—0.68 per gram; 20.3 per ounce.

MILK AND MILK PRODUCTS (*See* Accepted Foods, 1939, p. 230).

Denia S. A. Compania Anonima, Long Island City, N. Y.

DENIA, a spray-dried whole milk powder.

Analysis (submitted by manufacturer).—Moisture 2.0%, total solids 98.0%, ash 5.6%, protein (N × 6.38) 26.7%, fat (Mojonnier Method) 28.0%, lactose (by difference) 37.7%.

Calories.—5.10 per gram; 145 per ounce.

PREPARATIONS USED IN THE FEEDING OF INFANTS, FRUITS, VEGETABLES AND OTHER PREPARATIONS (*See* Accepted Foods, 1939, p. 185).

Libby, McNeill & Libby, Chicago.

LIBBY'S BRAND CHOPPED CARROTS.

Analysis (submitted by manufacturer).—Moisture 92.8%, total solids 7.2%, ash 1.0%, sodium chloride 0.7%, fat (ether extract) 0.1%, protein (N × 6.25) 0.7%, crude fiber 0.7%, carbohydrates other than crude fiber (by difference) 4.7%, calcium (Ca) 0.02%, phosphorus (P) 0.02%, iron (Fe) 0.0009%, copper (Cu) 0.00005%.

Calories.—0.22 per gram; 6.3 per ounce.

LIBBY'S BRAND CHOPPED BEETS.

Analysis (submitted by manufacturer).—Moisture 91.0%, total solids 9.0%, ash 1.2%, sodium chloride 0.8%, fat (ether extract) 0.01%, protein (N × 6.25) 0.8%, crude fiber 0.6%, carbohydrates other than crude fiber (by difference) 6.3%, calcium (Ca) 0.015%, phosphorus (P) 0.011%, iron (Fe) 0.0007%, copper (Cu) 0.00009%.

Calories.—0.29 per gram; 8.2 per ounce.

LIBBY'S BRAND CHOPPED GREEN BEANS.

Analysis (submitted by manufacturer).—Moisture 93.4%, total solids 6.6%, ash 0.9%, sodium chloride 0.7%, fat (ether extract) 0.04%, protein (N × 6.25) 1.2%, crude fiber 1.3%, carbohydrates other than crude fiber (by difference) 3.2%, calcium (Ca) 0.034%, phosphorus (P) 0.027%, iron (Fe) 0.0009%, copper (Cu) 0.0001%.

Calories.—0.18 per gram; 5.1 per ounce.

LIBBY'S BRAND CHOPPED SPINACH.

Analysis (submitted by manufacturer).—Moisture 94.2%, total solids 5.8%, ash 1.4%, sodium chloride 0.9%, fat (ether extract) 0.2%, protein (N × 6.25) 1.8%, crude fiber 0.9%, carbohydrates other than crude fiber (by difference) 1.5%, calcium (Ca) 0.11%, phosphorus (P) 0.3%, iron (Fe) 0.0018%, copper (Cu) 0.00004%.

Calories.—0.15 per gram; 4.3 per ounce.

LIBBY'S BRAND MEATLESS SOUP, a chopped mixture of celery, potatoes, peas, carrots, tomato juice, soy bean flour, barley and salt.

Analysis (submitted by manufacturer).—Moisture 82.3%, total solids 17.7%, ash 1.4%, sodium chloride 0.8%, fat (ether extract) 0.3%, protein (N × 6.25) 3.2%, crude fiber 1.3%, carbohydrates other than crude fiber (by difference) 11.5%, calcium (Ca) 0.02%, phosphorus (P) 0.07%, iron (Fe) 0.0025%, copper (Cu) 0.00014%.

Calories.—0.61 per gram; 17 per ounce.

LIBBY'S BRAND CHOPPED CHICKEN LIVER SOUP, a mixture of chopped carrots, celery, tomato juice, chicken livers, barley flour, salt and onions.

Analysis (submitted by manufacturer).—Moisture 90.5%, total solids 9.5%, ash 1.1%, sodium chloride 0.8%, fat (ether extract) 0.3%, protein (N × 6.25) 2.0%, crude fiber 0.5%, carbohydrates other than crude fiber (by difference) 5.6%, calcium (Ca) 0.018%, phosphorus (P) 0.040%, iron (Fe) 0.0030%, copper (Cu) 0.00008%.

Calories.—0.33 per gram; 9.4 per ounce.

LIBBY'S BRAND CHOPPED APRICOTS, sweetened.

Analysis (submitted by manufacturer).—Moisture 81.4%, total solids 18.7%, ash 0.6%, sodium chloride 0.02%, fat (ether extract) 0.02%, protein (N × 6.25) 0.7%, crude fiber 0.6%, carbohydrates other than crude fiber (by difference) 16.8%, calcium (Ca) 0.009%, phosphorus (P) 0.025%, iron (Fe) 0.0008%, copper (Cu) 0.00009%.

Calories.—0.70 per gram; 20 per ounce.

LIBBY'S BRAND CHOPPED PEACHES, sweetened.

Analysis (submitted by manufacturer).—Moisture 83.7%, total solids 16.3%, ash 0.4%, sodium chloride 0.01%, fat (ether extract) 0.01%, protein (N × 6.25) 0.5%, crude fiber 0.6%, carbohydrates other than crude fiber (by difference) 14.8%, calcium (Ca) 0.007%, phosphorus (P) 0.018%, iron (Fe) 0.0007%, copper (Cu) 0.00005%.

Calories.—0.61 per gram; 17 per ounce.

LIBBY'S BRAND CHOPPED PEARS, sweetened.

Analysis (submitted by manufacturer).—Moisture 82.9%, total solids 17.1%, ash 0.3%, sodium chloride 0.05%, fat (ether extract) 0.02%, protein (N × 6.25) 0.3%, crude fiber 1.2%, carbohydrates other than crude fiber (by difference) 15.3%, calcium (Ca) 0.010%, phosphorus (P) 0.014%, iron (Fe) 0.0016%, copper (Cu) 0.00013%.

Calories.—0.63 per gram; 18 per ounce.

LIBBY'S BRAND CHOPPED PRUNES WITH LEMON JUICE.

Analysis (submitted by manufacturer).—Moisture 67.5%, total solids 32.5%, ash 1.0%, sodium chloride 0.03%, fat (ether extract) 0.1%, protein (N × 6.25) 1.0%, crude fiber 1.8%, carbohydrates other than crude fiber (by difference) 28.6%, calcium (Ca) 0.031%, phosphorus (P) 0.039%, iron (Fe) 0.0051%, copper (Cu) 0.0001%.

Calories.—1.19 per gram; 34 per ounce.

PREPARATIONS USED IN THE FEEDING OF INFANTS (*See* Accepted Foods, 1939, p. 156).

Beech-Nut Packing Co., Canajoharie, N. Y.

BEECH-NUT BRAND CHOPPED VEGETABLES AND BEEF WITH RICE AND BARLEY, a cooked mixture of chopped carrots, potatoes, beef, celery, tomato puree, rice, barley and sodium chloride.

Analysis (submitted by manufacturer).—Moisture 85%, total solids 15%, ash 1.5%, fat (ether extract) 0.8%, protein (N × 6.25) 3.4%, crude fiber 0.5%, carbohydrates other than crude fiber (by difference) 8.8%, calcium (Ca) 0.02%, phosphorus (P) 0.031%, iron (total Fe) 0.001%, iron (available Fe)[1] 0.0007%, copper (Cu)[2] 0.00009%, specific gravity 1.044.

Calories.—0.56 per gram; 16 per ounce.

BEECH-NUT BRAND CHOPPED APRICOTS AND APPLESAUCE, a cooked mixture of chopped apricots (dried without sulfur dioxide) and apples.

Analysis (submitted by manufacturer).—Moisture 83%, total solids 17%, ash 0.6%, fat (ether extract) 0.1%, protein (N × 6.25) 0.6%, crude fiber 0.8%, sugars after inversion (as sucrose) 10.6%, carbohydrates other than crude fiber (by difference) 14.9%, calcium (Ca) 0.025%, phosphorus (P) 0.018%, iron (total Fe) 0.0008%, iron (available Fe)[1] 0.0007%, copper (Cu)[2] 0.00013%, specific gravity 1.078.

Calories.—0.63 per gram; 18 per ounce.

BEECH-NUT BRAND STRAINED APRICOTS AND APPLES, a cooked mixture of apricots (dried without sulfur dioxide) and apples.

Analysis (submitted by manufacturer).—Moisture 81.8%, total solids 18.2%, ash 0.7%, fat (ether extract) 0.1%, protein (N × 6.25) 0.6%, crude fiber 0.6%, sugars after inversion (as sucrose) 11.4%, carbohydrates other than crude fiber (by difference) 16.1%, calcium (Ca) 0.025%, phosphorus (P) 0.019%, iron (total Fe) 0.0008%, iron (available Fe)[1] 0.0007%, copper (Cu)[2] 0.00015%, specific gravity 1.077.

Calories.—0.68 per gram; 19 per ounce.

BEECH-NUT BRAND STRAINED SQUASH.

Analysis (submitted by manufacturer).—Moisture 89.7%, total solids 10.3%, ash 0.5%, fat (ether extract) 0.3%, protein (N × 6.25) 1.1%, crude fiber 0.1%, carbohydrates other than crude fiber (by difference) 8.3%, calcium (Ca) 0.03%, phosphorus (P) 0.012%, iron (total Fe) 0.0009%, iron (available Fe)[1] 0.0007%, copper (Cu) 0.0002%, specific gravity 1.053.

Calories.—0.4 per gram; 11 per ounce.

BEECH-NUT BRAND CHOPPED VEGETABLES AND LAMB WITH RICE, a chopped cooked mixture of carrots, potatoes, celery, onions, milk, lamb, lamb liver, rice and sodium chloride.

Analysis (submitted by manufacturer).—Moisture 85.3%, total solids 14.7%, ash 1.7%, fat (ether extract) 1.1%, protein (N × 6.25) 2.3%, crude fiber 0.4%, carbohydrates other than crude fiber (by difference) 9.2%, calcium (Ca) 0.036%, phosphorus (P) 0.047%, iron (total Fe) 0.00061%, iron (available Fe) 0.00059%, copper (Cu) 0.00008%.

Calories.—0.56 per gram; 16 per ounce.

SUGARS AND SYRUPS (*See* Accepted Foods, 1939, p. 324).

A. E. Staley Manufacturing Company, Decatur, Ill.

STALEY'S SWEETOSE BRAND GOLDEN SYRUP, a mixture of sweetose and refiners' syrup.

Analysis (submitted by manufacturer).—Moisture 25.5%, total solids 74.5%, ash (residue remaining at 500 to 550 C.) 0.6%, reducing sugars as dextrose 48.0%, sucrose 4.0%, dextrins (nonfermentable) 22.3%, total carbohydrates (by difference) 73.4%.

Calories.—3.0 per gram; 85 per ounce.

VEGETABLES AND MUSHROOMS (*See* Accepted Foods, 1939, p. 340).

General Foods Corporation, New York, product distributed by Frosted Foods Sales Corporation, New York.

BIRDS EYE BRAND QUICK-FROZEN SPINACH, quick-frozen, ready-to-cook spinach.

Analysis (submitted by manufacturer).—Average: Moisture 91.1%, total solids 8.9%, ash 1.3%, fat (ether extract) 0.5%, protein (N × 6.25) 2.9%, invert sugar 0.2%, sucrose 0.5%, crude fiber 0.7%, carbohydrates other than crude fiber (by difference) 3.5%. Range: Moisture 89.6 to 91.6%, total solids 8.4 to 10.4%, ash 1.2 to 1.4%, fat (ether extract) 0.4 to 0.6%, protein (N × 6.25) 2.4 to 3.3%, sucrose 0.1 to 1.2%, invert sugar 0.1 to 0.3%, crude fiber 0.7 to 0.8%, carbohydrates other than crude fiber (by difference) 2.8 to 4.5%.

The firm reports (1941) that the product contains (fresh basis) 300 international units of vitamin A per gram,[3] 3.4 international units of thiamine per gram,[4] 1.1 Sherman-Bourquin units of riboflavin per gram,[4] and 6.6 international units of vitamin C per gram.[3]

Calories.—Average: 3 per gram; 85 per ounce. Range: 2.8 to 3.8 per gram; 79 to 108 per ounce.

---

1. According to Shackleton and McCance: The Ionizable Iron in Foods, Biochem. J. **30:** 582 and Hill: A Method for the Estimation of Iron in Biological Materials, Proc. Roy Soc. London. Series B **107:** 205, 1930.

2. According to method of McFarlane, W. D.: Application of the Sodium Diethyl Dithiocarbamate Reaction to the Micro-Colorimetric Determination of Copper in Organic Substances, Biochem. J. **26:** 1022, 1932.

3. Vitamin A was calculated on the basis that 0.6 microgram of carotene equals 1 international unit. Fitzgerald, G. A., and Fellers, C. R.: Carotene and Ascorbic Acid Content of Fresh Market and Commercially Frozen Fruits and Vegetables, Food Research **3:** 109, 1938.

4. Fellers, C. R.; Esselen, W. B., Jr., and Fitzgerald, G. A.: Vitamin B_1 and Vitamin B_2 (G) Content of Vegetables as Influenced by Quick Freezing and Canning, Food Research **5:** 495, 1940.

THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION

535 NORTH DEARBORN STREET - - - CHICAGO, ILL.

Cable Address - - - - "Medic, Chicago"

Subscription price - - - - - Eight dollars per annum in advance

Please send in promptly notice of change of address, giving both old and new; always state whether the change is temporary or permanent. Such notice should mention all journals received from this office. Important information regarding contributions will be found on second advertising page following reading matter.

SATURDAY, AUGUST 2, 1941

WAR AND THE SPREAD OF EPIDEMIC DISEASES

Again the four horsemen of the apocalypse are riding. Once more war, hunger, disease and death destroy mankind. The horsemen of today travel with the increased speed of mechanized transportation. Today the possibilities for sudden and widespread outbreaks of disease arising far from their endemic foci are more likely than ever previously.

Among the endemic diseases which are kept from epidemic proportions only by the most stringent public health activities are malaria, yellow fever, typhus and plague. News dispatches from Europe indicate already that typhus is being held in check only with the greatest difficulty. Indeed, with the fragmentary information available it has probably reached serious epidemic proportions already in certain areas, especially Poland and the Balkans.

No doubt plague, as far as this country is concerned, is a problem of greater potentiality. Epidemiologic data give definite information on the comparative permanence of endemic foci of plague infections in localities many years after epidemics of the disease have subsided. Once plague has appeared in a community, especially in cities with extensive port facilities, all known control measures for this disease must remain actively employed by the local public health agencies for prolonged periods. There can be no cessation of control measures. These demand the careful checking of the infectivity of fleas combed from rats and other rodents. This is done by the inoculation of selected and area-notated pools of fleas into guinea pigs, and the subsequent identification of the plague bacillus by suitable culture methods when evidence of the disease occurs in the guinea pigs.

Although atmospheric conditions are still an obscure factor in the spread and periodic acceleration in virulence of many diseases, in plague relative high humidity and low and equitable mean temperatures seem to play an important part. Moreover, it is possible in areas in which the infection has remained subdued or dormant that atypical unrecognized lesions on necropsy may occur in rats; perhaps some of these rodents may be healthy carriers. Under such circumstances, inoculations of pools of fleas may become an important source of information.

Reported plague infection has been recently proved in fleas from rats in San Francisco, from ground squirrels in Kern County, California, from ground squirrels in Ada County, Idaho, and from a marmot in Malheur County, Oregon.[1]

In San Francisco County the health department, with the assistance of the United States Public Health Service, has maintained a system of plague control since 1908, consisting of the enforcement of ratproof ordinances in buildings, trapping, poisoning and examinations of the carcasses of rodents by necropsy, laboratory cultures when necessary, and, finally, in 1938, inoculation of pools of fleas into guinea pigs.

In spite of the fact that the rat has always been regarded as the source of epidemics in human beings, the discovery of widespread distribution of plague in the ground squirrel and perhaps other rodents is not reassuring. To control this serious menace there must be a careful integration of the control activities of cities, counties, states and the federal government, with the use of trained personnel and the appropriation of adequate funds. If this is not done, the consequences may be indeed tragic.

Clearly the insulation of this country from the disease consequences of war will prove a colossal task and will require the most careful planning and effort.

AUROTHERAPY IN RHEUMATOID ARTHRITIS

Previous to 1936, reports on the use of gold salts in the treatment of arthritis had appeared mainly in European and British journals. In 1935 an editorial appeared in THE JOURNAL[1] surveying some of these reports and warning of the attendant dangers of gold therapy. Since that date communications have appeared not infrequently in domestic scientific literature. At a meeting of the American Rheumatism Association[2] a physician stated that 976 out of 3,229 articles on arthritis listed in the *Quarterly Cumulative Index Medicus* from 1932 through 1936 dealt solely with treatment; 80 of the 976 discussed aurotherapy. The majority of communications pointedly draw attention to toxic possibilities. The more enthusiastic investigators are convinced that clinical results offset the untoward reactions; the more conservative advise caution and further experimentation and clinical investigation before deciding on the efficacy of gold therapy in arthritis.

1. Plague Infection in California, Idaho and Oregon, Pub. Health Rep. **56**: 1295 (June) 20) 1940.

1. Gold Therapy in Arthritis, editorial, J. A. M. A. **105**: 2163 (Dec. 28) 1935.

2. Snyder, R. G.; Lust, F. J.; Traeger, C. H., and Kelly, L. C.: Gold Salt Therapy in Chronic Arthritis, abstr. J. A. M. A. **109**: 1307 (Oct. 16) 1937.

Inquiries which come to THE JOURNAL and to the Council on Pharmacy and Chemistry indicate that promotional activities for the use of gold preparations are not quiescent. Gold preparations have been offered for oral, intramuscular, intravenous and topical use, the latter as a nasal instillant, gargle, paste, cream or suspension. They have been advocated for rheumatoid arthritis and osteoarthritis, septicemia, typhoid, bronchopneumonia, asthma, influenza, pulmonary tuberculosis, epidemic encephalitis, alcoholism, migraine and malnutrition. Again, they have been suggested by some pharmaceutic houses as sedatives, antispasmodics, analgesics, heart stimulants, hematinics, to treat debility and neurosis and even for acute and chronic nephritis. An example of the exaggerated and misleading claims not infrequently encountered is cited in a report [3] by the Council on Pharmacy and Chemistry. This report relates to a colloidal gold preparation promoted for therapeutic use in the treatment of cancer.

The dangers of chrysotherapy make it evident that this type of therapy should be undertaken only by those who have had extensive experience and who are fully aware of the dangers. Untoward reactions have been discussed before in THE JOURNAL.[4] Those reported include generalized erythema, dermatitis, chrysiasis, granulocytopenia, aplastic anemia, purpura hemorrhagica, alopecia, stomatitis, ulceration of the mouth, stomach and upper part of the intestine, enteritis, nausea and vomiting, bronchial irritation, fever, generalized pruritus, deafness, delirium, hepatic impairment, edema of the glottis, labial edema, hyperkeratosis of the soles, herpes zoster, lichen planus, erythema nodosum, nephritis, prostration and death. This is an imposing list of possible toxic manifestations for a form of treatment that is the object of renewed attention. Periodically, gold therapy for various pathologic processes has been revived, but, without exception, interest has decreased when reports of intoxication came to light. The Council on Pharmacy and Chemistry recognizes the use of some gold preparations only in the therapy of lupus erythematosus.

The Council recently reviewed the recent publications on chrysotherapy in rheumatoid arthritis. These reports appeared to indicate that, when careful histories were taken, reactions ranging from a mild to a severe character occurred in almost half of the treated patients. As regards the therapeutic outcome, the percentage of reported "cures" is appreciably lower in the United States than in foreign countries. Again, it has been stated [5] that great relief has been obtained in only 10 to 35 per cent of the cases, according to American reports, contrasting with 50 to 70 per cent reported in foreign publications.

Do the results warrant the risks? Many European workers believe that gold salts for rheumatoid arthritis are worthy of trial; but the majority of the more cautious American investigators adopt a conservative attitude about more general employment of aurotherapy. In the opinion of the Council on Pharmacy and Chemistry there do not appear to be sufficient controlled data to warrant recognition of the use of gold salts for other than selected cases of lupus erythematosus. The precise way in which gold acts is not known, and complete success with this form of therapy may be considered highly problematic. Again, a careful search for possible contraindications must always be made prior to institution of treatment. In view of the dangerous possibilities, the definite place of aurotherapy in rheumatoid arthritis remains controversial. Certainly the use of gold is always accompanied by the possibility of untoward reactions, and these reactions have terminated more than once in the death of the patient.

3. Collodaurum Not Acceptable for N. N. R., Reports of the Council on Pharmacy and Chemistry, J. A. M. A. **111**: 531 (Aug. 6) 1938.

4. Anderson, N. L., and Palmer, W. L.: The Danger of Gold Salt Therapy, J. A. M. A. **115**: 1627 (Nov. 9) 1940. Perry, M. W.: Gold Injections and Colitis, ibid. **113**: 965 (Sept. 2) 1939. Margolis, H. M., and Eisenstein, V. W.: Some Specific Measures in the Treatment of Rheumatoid Arthritis, ibid. **114**: 1429 (April 13) 1940. Gold Therapy in Arthritis.[1] Gold Sodium Thiosulfate in Arthritis.[2]

5. Gold Sodium Thiosulfate in Arthritis, Queries and Minor Notes, J. A. M. A. **114**: 2049 (May 18) 1940.

Current Comment

RETURN YOUR INFORMATION CARD FOR THE DIRECTORY PROMPTLY

About September 1 an information card will be sent from the headquarters office of the American Medical Association to every physician in the United States and Canada. The information secured is to be used in compiling the Seventeenth Edition of the American Medical Directory. The directory is prepared at regular intervals in the Biographic Department of the American Medical Association. The last previous edition appeared in 1940. This volume is one of the most important contributions of the American Medical Association to the work of the medical profession in the United States; it has been especially valuable in the medical preparedness program. In it, as in no other published directory, are dependable data concerning physicians, hospitals, medical organizations and activities. The directory provides full information concerning medical colleges, specialization in the field of medical practice, memberships in special medical societies, tabulations of medical journals and medical libraries and, indeed, practically every important fact concerning the medical profession in which any one might possibly be interested. Before filling out the information card, read the instructions carefully. Physicians are especially urged to state whether or not they are on extended active duty for the medical reserve corps of the United States Army and Navy. Fill out the card and return it promptly whether or not a change has occurred in any points on which information is requested. If a change of address occurs before March 1, 1942 report it at once. Should you fail to receive a card before the first of October, write at once to the headquarters office stating that fact and indicating such changes as you may desire in the information published in the 1940 issue of the directory.

NASAL OBSTRUCTION AND IMPAIRMENT OF HEARING

Literature contains many theories as to the causation of chronic types of impairment of hearing. Many otologists believe that nasal obstruction causes or contributes to certain types of deficiency of hearing and advocate submucous resection of the nasal septum as treatment for chronic nonsuppurative otitis media. Apparently the only way in which nasal obstruction could interfere with hearing is by causing some involvement of the eustachian tube, with subsequent impairment of the ventilation of the middle ear. The Report of the Committee on the Problems of the Hard of Hearing from the Section on Laryngology, Otology and Rhinology of the American Medical Association [1] states that "the most important etiologic factors according to the majority of otologists are nose and throat conditions that directly affect the eustachian tube." In children the most potent cause of tubal inflammation is adenoids. About 85 per cent of all deafness is due to middle ear involvement, and 90 per cent of these middle ear involvements have their origin in inflammatory conditions of the nasopharynx with extension to the ear by way of the eustachian tube. In a recent article Johnson [2] deprecates the tendency to associate middle ear disease etiologically with anatomic variations in the nasal chambers, a theory based on the supposition that these variations cause middle ear disease by mechanically altering the air current in the nasopharynx. Shambaugh's [3] 2 cases of congenital atresia of the choanae with complete blockage of the air passage to the nasopharynx and without middle ear disease disprove this contention. A common cause of disturbance of sound conduction is to be seen in the occlusion of the pharyngeal end of the eustachian tube by adenoids, tumor, hyperplasia of the posterior end of the turbinals, and common inflammations such as colds. Johnson believes that local noninflammatory disease of the nose, such as deformity of the septum, cannot lead to occlusion of the tubes and therefore cannot be a cause of difficulty with hearing. Shambaugh [4] points out that the various operations on the nose for the relief of ear trouble are based on the erroneous confusion of the tubotympanic processes, so common in childhood, with the chronic persisting processes which occur in adult life. Crowe and Baylor [5] found an abnormal growth of lymphoid tissue partially obstructing the nasopharyngeal orifice of the eustachian tube in children with impaired hearing for high tones. They treated this lymphoid hyperplasia by means of a radium bomb and obtained excellent results. They state that if the condition is treated before the age of 15 the hearing usually returns to near the normal level; after this age the lesions in the middle ear may have progressed to the point of being irreversible. Johnson performed submucous resection for nasal obstruction in 46 cases. In 35 (65 per cent) of these there was either no deficiency in hearing or only high tonal dips. In the cases in which some impairment of hearing was present, the improvement demonstrated with the audiometer was too insignificant to warrant urging the operation as a means of improving hearing. Johnson concludes that nasal obstruction does not cause a characteristic impairment of hearing and that therefore submucous resection is not to be recommended as a means of improving deficiency in hearing.

1. Hays, Harold; Newhart, Horace; Pierce, Norval; Phillips, Wendell, and Shambaugh, George: Report of the Committee on the Problems of the Hard of Hearing, Section on Laryngology, Otology and Rhinology, J. A. M. A. **83:** 2094 (Dec. 27) 1924.

2. Johnson, M. R.: Nasal Obstruction and Impairment of Hearing: Report of Forty-Six Cases of Submucous Resection with Audiometric Studies, Arch. Otolaryng. **33:** 536 (April) 1941.

3. Shambaugh, G. E.: The Relation of Nose and Throat to Ear Diseases, Illinois M. J. **42:** 431 (Dec.) 1922.

4. Shambaugh, G. E.: Facts and Fancies in the Practice of Otolaryngology, J. A. M. A. **87:** 1720 (Nov. 20) 1926.

5. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112:** 585 (Feb. 18) 1939.

DARK ADAPTATION AND VITAMIN A

The use of dark adaptation tests as means of determining vitamin A deficiency has received much study. Recently Jeans and his colleagues [1] have presented further evidence that the biophotometer, which is one of the instruments used for this purpose, gives consistent testing results with regard to the vitamin A status of human beings. Normal subjects with normal vitamin A intake, while remaining in good health, have normal test results with this instrument at all times. The subjects studied by Jeans and his co-workers who had poor dark adaptation and who submitted to therapy obtained normal adaptation in all instances while receiving vitamin A. In the geographic region in which the tests were made, dietary deficiencies alone seemed to be the cause of impaired dark adaptation in but few instances. The most frequent and usual cause of impaired adaptation seemed to be a utilization deficiency resulting from illness, especially when due to infection. In an orphanage in which children were receiving approximately 2,000 units of vitamin A in the daily diet, instances of abnormal dark adaptation by the biophotometer test were not observed during a winter period of observation. In another group of one hundred and twenty school children, however, 5 per cent had dark adaptation not wholly normal in the fall and almost 20 per cent demonstrated abnormal adaptation in the winter. This variation in seasonal incidence, these investigators believe, is related more closely to the seasonal occurrence of infection than to seasonal variations in diet. In a study comparing the biophotometer and the Hecht-Schlaer adaptometer as comparable instruments for measuring dark adaptation, Eckardt and Johnson [2] found that the rod adaptation as measured with the biophotometer and the Hecht-Schlaer adaptometer is identical, although some difficulty may be encountered in measuring the cone adaptation with the biophotometer. They feel, however, that the continued use of the biophotometer in the study of vitamin A deficiency is valid and gives a true picture of dark adaptation. Although the problems of measuring vitamin A deficiency by means of dark adaptation tests are complex and somewhat controversial, the evidence at hand indicates that some of these tests, at least when carefully controlled, give sufficiently consistent results, at least by competent observers, to warrant their acceptance as accurate measures.

1. Jeans, P. C.; Blanchard, Evelyn L., and Satterthwaite, F. E.: Dark Adaptation and Vitamin A: Further Studies with the Biophotometer, J. Pediat. **18:** 170 (Feb.) 1941.

2. Eckardt, R. E., and Johnson, L. V.: A Comparison of Two Methods of Measuring Dark Adaptations, J. Pediat. **18:** 195 (Feb.) 1941.

MEDICAL PREPAREDNESS

In this section of The Journal each week will appear official notices by the Committee on Medical Preparedness of the American Medical Association, announcements by the Surgeon Generals of the Army, Navy and Public Health Service, and other governmental agencies dealing with medical preparedness, and such other information and announcements as will be useful to the medical profession.

NATIONAL GUARD MEDICAL OFFICERS ON ACTIVE DUTY

Following is a list of the medical officers on duty with National Guard units of the various states which have been called into active service, together with the organization and camp at which they were serving when this report was recently submitted to these headquarters. Those from the other states were published last week.

NEW YORK

ADAMS, Vincent J., 1st Lieut., Brooklyn, 186th Field Artillery, Madison Barracks, Sackets Harbor, N. Y.
AINSWORTH, Thomas H., Captain, Beacon, 156th Field Artillery.
BACCHIANI, Torquato L., 1st Lieut., Nesconset, 101st Signal Battalion, Camp Shelby, Miss.
BALL, Thomas L., 1st Lieut., New York City, 187th Field Artillery, Fort Ethan Allen, Vt.
BOYD, James R., Major, Brooklyn, 245th Coast Artillery (HD), Fort Hancock, N. J.
CALHOUN, Douglas A., Captain, Ray Brook, 105th Infantry.
CARRANO, Armand T., 1st Lieut., Brooklyn, 187th Field Artillery, Fort Ethan Allen, Vt.
CARTER, Robert E., Major, Jamaica, 369th Coast Artillery (AA), Fort Ontario, Oswego, N. Y.
CERAVOLO, Raphael J., 1st Lieut., Brooklyn, 186th Field Artillery, Madison Barracks, Sackets Harbor, N. Y.
CLARK, George B., Major, Armonk, 108th Inf., Fort McClellan, Ala.
COLE, Robert F., 1st Lieut., Brooklyn, 102d Med. Regt., Fort McClellan.
CONSTANTINO, Joseph G., 1st Lieut., Flushing, 101st Cavalry.
CRATER, Robert L., Captain, New York City, 369th Coast Artillery (AA), Fort Ontario, Oswego, N. Y.
DAVIS, Ralph H., Major, Penn Yan, 134th Med. Regt., Fort Bragg.
DINERMAN, Benjamin V., Captain, Brooklyn, 101st MP Battalion.
DUNGAN, Clarence E., 1st Lieut., Auburn, 134th Med. Regt., Ft. Bragg.
EPSTEIN, William M., 1st Lieut., Newark, N. J., 134th Medical Regiment, Fort Bragg, N. C.
EVANS, John A., Captain, New York City, 207th Coast Artillery (AA), Camp Stewart, Ga.
FITZGERALD, Thomas G., 1st Lieut., Albany, 134th Medical Regiment, Fort Bragg, N. C.
FUGE, Wilfred W., Captain, Buffalo, 174th Infantry, Fort Dix, N. J.
GAUS, Louis H., Colonel, Ticonderoga, State Staff.
GOLDSTEIN, Philip, Captain, Brooklyn, 105th Field Artillery, Fort McClellan, Ala.
GORSCH, Rudolph V., Major, New York City, 207th Coast Artillery (AA), Camp Stewart, Ga.
GRIFFIN, Donald C., Major, New York City, 71st Infantry.
HAMILL, John A. C., Captain, Port Chester, 102d Medical Regiment, Fort McClellan, Ala.
HANLON, Lawrence W., Capt., Port Chester, 209th Coast Artil., (AA).
HAYMAN, Charles R., Captain, White Plains, 258th Field Artillery, Fort Ethan Allen, Vt.
HEBEL, Herbert D., 1st Lieut., Auburn, 134th Med. Regt., Fort Bragg.
HOEY, Patrick H., 1st Lieut., Scarsdale, 102d Medical Regiment, Fort McClellan, Ala.
HOOD, Robert I., Captain, Corning, 134th Med. Regt., Fort Bragg, S. C.
HUBER, Henry S., Captain, New York City, 207th Coast Artillery (AA), Camp Stewart, Ga.
JACKSON, William O., Major, Brooklyn, 102d Medical Regiment, Fort McClellan, Ala.
JOHNSON, Harry G., Major, Buffalo, 174th Infantry, Fort Dix, N. J.
JOHNSON, Paul C., 1st Lieut., Penn Yan, 134th Med. Regt., Fort Bragg.
JOHNSON, Ted, Capt., N. Y. City, 102d Med. Regt., Fort McClellan.
JONES, Ceirianog H., 1st Lieut., Scranton, Pa., 244th Coast Artillery, Camp Pendleton, Va.
KALLET, Joseph R., 1st Lieut., Syracuse, 108th Inf., Fort McClellan.
KELLEHER, Vincent R., 1st Lieut., Fort Edward, 134th Medical Regiment, Fort Bragg, N. C.
LANE, Theron I., Captain, Ossining, 244th Coast Artillery, Camp Pendleton, Va.
LASKY, Mortimer A., Major, Brooklyn, 186th Field Artillery, Madison Barracks, Sackets Harbor, N. Y.
LEFKOWITZ, Michael P., 1st Lieut., New York City, 105th Field Artillery, Fort McClellan, Ala.
LUBITZ, Benjamin, Major, Brooklyn, 102d Regiment, Fort McClellan.
MacDONALD, John K., Major, White Plains, 102d Medical Regiment, Fort McClellan, Ala.
MANDRACCHIA, John L., Major, New York City, 212th Coast Artillery (AA).
MARCOU, Milton, 1st Lieut., Brooklyn, 105th Field Artillery, Fort McClellan, Ala.
McCASTOR, Joseph T. N., Captain, New York City, 102d Engineers, Fort McClellan, Ala.
McDONOUGH, Thomas C., 1st Lieut., Buffalo, 102d Separate Battalion Coast Artillery (AA).
McKENNA, Walter D., Major, Troy, 105th Infantry.
McKEON, John G., 1st Lieut., Albany, 134th Med. Regt., Fort Bragg.
McLAUGHLIN, John J., 1st Lieut., Albany, 101st Anti-Tank Battalion, Fort Benning, Ga.
MIERAS, Marion D., 1st Lieut., Elmira, 134th Med. Regt., Fort Bragg.
MOORE, Francis W., Colonel, Brooklyn, 134th Med. Regt., Fort Bragg.
MURPHY, James M., 1st Lieut., Willard, 134th Med. Regt., Fort Bragg.
MURRAY, William J., 1st Lieut., Binghamton, 106th Infantry, Fort McClellan, Ala.
NILES, Charles E., Major, Poughkeepsie, 155th Field Artillery.
NORMAN, Abraham, Captain, New York City, Special Troops, 27th Division, Fort McClellan, Ala.
NORTHRIDGE, John A., Captain, Brooklyn, 102d Medical Regiment, Fort McClellan, Ala.
O'BRIEN, Richard A., Major, Corning, 134th Med. Regt., Fort Bragg.
OEHLER, Harry R., 1st Lieut., Buffalo, 174th Infantry, Fort Dix, N. J.
OLSHANSKY, Abraham L., Major, Albany, 106th Inf., Fort McClellan.
PAGANELLI, Americus J., Captain, New York City, 245th Coast Artillery, Fort Hancock, N. J.
PAGANELLI, Joseph E., Major, New York City, 244th Coast Artillery, Camp Pendleton, Va.
PIERCE, Lee R., Lieut. Colonel, New York City, 102d Medical Regiment, Fort McClellan, Ala.
POMERANZE, Julius, 1st Lieut., New York City, 102d Quartermaster Regiment, Fort McClellan, Ala.
POST, Arthur, Capt., N. Y. City, 258th Fld. Artillery, Ft. Ethan Allen.
PRISCO, Frank J., 1st Lieut., New York City, 102d Medical Regiment, Fort McClellan, Ala.
ROGERS, William K., Major, Flushing, 101st Cavalry.
ROONEY, James F., Lieut. Colonel, Albany, 134th Medical Regiment, Fort Bragg, N. C.
RUSSELL, Nelson Gorham, Jr., 1st Lieut., Buffalo, 174th Inf., Fort Dix.
SAFIAN, Harold, 1st Lieut., New York City, 102d Medical Regiment, Fort McClellan, Ala.
SARGENT, Carlton W., 1st Lieut., Dundee, 134th Med. Regt., Ft. Bragg.
SAUNDERS, William, 1st Lieut., Troy, 106th Inf., Fort McClellan, Ala.
SCHIMENTI, Matthew L., 1st Lieut., Yonkers, 165th Infantry, Fort McClellan, Ala.
SCHINTZIUS, William C., Capt., Boonville, 106th Inf., Fort McClellan.
SCHUTKEKER, Bruno G., Captain, Buffalo, 174th Inf., Fort Dix, N. J.
SIMON, Kona, 1st Lieut., New York City, 101st MP Battalion.
SMITH, DeWitt H., 1st Lieut., New York City, 102d Medical Regiment, Fort McClellan, Ala.
SNOW, Herman B., Captain, Binghamton, 106th Inf., Fort McClellan.
SNYDER, Charles T., Major, New York City, 167th Field Artillery, Fort Ethan Allen, Vt.
SOMMER, Abram E., Major, Brooklyn, 102d Quartermaster Regiment, Fort McClellan, Ala.
STAGNITTO, Rosario J., 1st Lieut., Rochester, 209th Coast Artil. (AA).
STOCKHAMMER, Raymond J., Captain, New York City, 165th Infantry, Fort McClellan, Ala.
STOCKHAMMER, Stanley F., Captain, Forest Hills, 165th Infantry, Fort McClellan, Ala.
TAYLOR, William G., Captain, Buffalo, 209th Coast Artillery (AA).
TERRY, Richard N., 1st Lieut., Buffalo, 174th Infantry, Fort Dix, N. J.
THOMSON, John D., Captain, Syracuse, 108th Inf., Fort McClellan.
TURIGA, John R., Captain, Beacon, 102d Med. Regt., Fort McClellan.
UNDERWOOD, Edward B., Major, Brooklyn, 102d Medical Regiment, Fort McClellan, Ala.
VELARDI, Martin J., Captain, Brooklyn, 187th Field Artillery, Fort Ethan Allen, Vt.
WARD, Harrison F., Major, Rochester, 102d Med. Regt., Fort McClellan.
WARREN, Arthur F., Capt., N. Y. City, 102d Obsn. Sq., Fort McClellan.
WEED, Jefferson, Captain, Garden City, 102d Anti-Tank Battalion.
WELKER, George, Jr., 1st Lieut., Garden City, 212th Coast Artil. (AA).
WELLING, Joseph G., Captain, Long Beach, 102d Medical Regiment, Fort McClellan, Ala.
WENDELKEN, Herbert F., 1st Lieut., Brooklyn, 245th Coast Artillery, Fort Hancock, N. J.
ZITTEL, Harold E., Major, Buffalo, 106th Field Artillery.

NORTH CAROLINA

BREWTON, William A., Captain, Enka, 105th Engineers, Fort Jackson.
CARTER, Paul C., Major, Madison, 105th Med. Regt., Fort Jackson.

COX, Alexander M., Capt., Madison, 105th Med. Regt., Fort Jackson.
CRAVEN, Thomas, Major, Huntersville, 105th Engineers, Fort Jackson.
FENNER, Edwin F., Lieut. Colonel, Henderson, 105th Medical Regiment, Fort Jackson, S. C.
FLEMING, Frank R., Capt., Statesville, 105th Med. Regt., Fort Jackson.
FOX, Norman A., Captain, Greensboro, 252d Coast Artillery, Fort Moultrie, S. C.
GLASCOCK, Harold W., Major, Raleigh, 105th Medical Regiment, Fort Jackson, S. C.
GOLEY, Willard C., Major, Graham, 120th Infantry.
MATHESON, Robert A., Major, Raeford, 252d Coast Artillery, Fort Moultrie, S. C.
MOORE, Henry B., Captain, Graham, 120th Infantry.
NEAL, Joseph W., Jr., 1st Lieut., Walnut Cove, 105th Medical Regiment, Fort Jackson, S. C.
PATTERSON, Fred G., Captain, Chapel Hill, 113th Field Artillery, Fort Jackson, S. C.
POWELL, Eppie C., Jr., Captain, Columbia, S. C., 113th Field Artillery, Fort Jackson, S. C.
REYNOLDS, Ernest H., Captain, Columbia, S. C., 105th Medical Regiment, Fort Jackson, S. C.
RODMAN, Robert B., Major, Wilmington, 105th Medical Regiment, Fort Jackson, S. C.
SULLIVAN, Victor T., Captain, Wilmington, 252d Coast Artillery, Fort Moultrie, S. C.
TAYLOR, Thomas J., Captain, Roanoke Rapids, 105th Medical Regiment, Fort Jackson, S. C.
WILKINSON, Charles T., Major, Wake Forest, 113th Field Artillery, Fort Jackson, S. C.
WISELY, Martin R., Capt., Edenton, 105th Med. Regt., Fort Jackson.
YOUNG, Wiley R., 1st Lieut., Angier, 120th Infantry.

NORTH DAKOTA

FLANNERY, Hubert F., 1st Lieut., Bottineau, 164th Infantry, Camp Claiborne, La.
FRANKLIN, Earl A., Capt., Gilby, 164th Infantry, Camp Claiborne, La.
GRIFFIN, Vernon M., Captain, Grand Forks, 188th Field Artillery, Fort Francis E. Warren, Wyo.
HAYNES, George H., Major, Lisbon, 188th Field Artillery, Fort Warren.
NUESSLE, Robert F., Captain, Bismarck, 188th Field Artillery, Fort Francis E. Warren, Wyo.
ROBERTSON, Frank O., Captain, East Grand Forks, Minn., 188th Field Artillery, Fort Francis E. Warren, Wyo.
SCHATZ, George, Captain, West Fargo, 164th Inf., Camp Claiborne, La.

OHIO

AKERS, Robert H., 1st Lieut., Stoutsville, 112th Medical Regiment, Camp Shelby, Miss.
BEACH, Wilber E., Major, Middle Point, 112th Medical Regiment, Camp Shelby, Miss.
BERLIN, Frederick P., Captain, Wapakoneta, 136th Field Artillery, Camp Shelby, Miss.
BILL, Herbert E., Capt., Cleveland, 107th Cavalry, Camp Forrest, Tenn.
BLINN, John C., Jr., Captain, New Philadelphia, 112th Medical Regiment, Camp Shelby, Miss.
BRETTELL, Howard W., Captain, Steubenville, 151st Medical Battalion, Fort McClellan, Ala.
BRIOLA, Patsie F., Captain, Cleveland, 145th Inf., Camp Shelby, Miss.
BURROUGHS, Shepard A., Capt., Ashtabula, 107th Cav., Camp Forrest.
CALDWELL, Harry E., Major, Delaware, 166th Inf., Camp Shelby, Miss.
CATON, Russell J., Major, Bucyrus, 135th Field Artillery, Camp Shelby.
CHRISTIANSEN, James N., Major, Cincinnati, 147th Inf., Camp Shelby.
COOPER, Wellington W., Major, Columbus, 372d Infantry.
CRAWFORD, Henry S., Capt., Cleveland, 112th Observation Squadron.
CRIST, Jerry O., Major, Centerburg, 112th Med. Regt., Camp Shelby.
CURTISS, Edgar J., Major, Lima, 112th Med. Regt., Camp Shelby, Miss.
DIX, Carr E., Capt., Columbus, 112th Med. Regt., Camp Shelby, Miss.
DORR, Hugh C., 1st Lieut., Columbus, 112th Med. Regt., Camp Shelby.
ENSIGN, Paul R., Lieut. Colonel, Toledo, 112th Medical Regiment, Camp Shelby, Miss.
FRIDLINE, Gaylord D., Capt., Ashland, 112th Med. Regt., Camp Shelby.
FRIEDMAN, Norman, 1st Lieut., Longacre, W. Va., 145th Infantry, Camp Shelby, Miss.
GALLEN, John J., Capt., Columbus, 134th Field Artillery, Camp Shelby.
GROOM, Horace E., Lieut. Colonel, Akron, 151st Medical Battalion, Fort McClellan, Ala.
GROTHJAN, Cletus A., Major, Toledo, 148th Infantry.
HOISTON, Guilford B., Captain, Columbus, 372d Infantry.
HOWE, Charles E., Capt., Westerville, 151st Med. Bat., Fort McClellan.
HUMPHREY, Harold L., Captain, Cincinnati, 147th Inf., Camp Shelby.
INMAN, James G., Captain, Manchester, 147th Inf., Camp Shelby, Miss.
JACKSON, Harry D., Colonel, Circleville, 112th Medical Regiment, Camp Shelby, Miss.
JOHNSTON, Albert M., Captain, Marysville, 166th Inf., Camp Shelby.
KAYLOR, Frederick W., 1st Lieut., Bellefontaine, 113th Medical Regt.
KOLB, Thomas V., Captain, South Charleston, 112th Medical Regiment, Camp Shelby, Miss.
LECHNER, Robert H., Captain, Berea, 151st Med. Bat., Fort McClellan.
MAY, James R., Captain, Cleveland, 145th Infantry, Camp Shelby, Miss.
McNERNEY, Neville H., Major, Cleveland, 145th Inf., Camp Shelby.
MICHAEL, Nicholas, Capt., Columbus, 112th Med. Regt., Camp Shelby.
MIKESELL, Hobart L., Major, West Liberty, 112th Medical Regiment, Camp Shelby, Miss.
MORSE, George D., Captain, Columbus, 166th Inf., Camp Shelby, Miss.
PITKIN, York N., Major, Cleveland, 151st Med. Bat., Fort McClellan.
REEVES, James R., Capt., Columbus, 134th Fld. Artillery, Camp Shelby.
RIDGEWAY, Joseph A., Captain, Columbus, Special Troops, 37th Division, Camp Shelby, Miss.
RUPPERSBERG, Anthony, Jr., Major, Bexley, 136th Field Artillery, Camp Shelby, Miss.
SACKS, Leon J., 1st Lieut., Akron, 151st Med. Bat., Fort McClellan.
SCOTT, Robert P., Jr., 1st Lieut., Hattiesburg, Miss., 145th Infantry, Camp Shelby, Miss.
SMILEY, Bernard, Captain, Scranton, Pa., 151st Medical Battalion, Fort McClellan, Ala.
SMILEY, John T., Captain, Akron, 151st Med. Bat., Fort McClellan, Ala.
SMITH, Kenneth D., Captain, Columbus, 166th Infantry, Camp Shelby.
SPANGLER, Frederick E., Captain, Somerset, 151st Medical Battalion, Fort McClellan, Ala.
STOUGHTON, Wilbur A., Captain, Columbus, 134th Field Artillery, Camp Shelby, Miss.
TAYLOR, Russell E., Capt., Sandusky, 151st Med. Bat., Fort McClellan.
TROUP, Paul, Captain, Dayton, 136th Fld. Artillery, Camp Shelby, Miss.
VOORHIS, Charles C., 1st Lieut., Cleveland, 107th Cavalry, Camp Forrest, Tenn.
WALKER, Winston F., Major, Rocky River, 112th Engineers.
WILSON, Sloan J., Major, Columbus, 112th Quartermaster Regiment.
YAW, Owen F., Captain, Cincinnati, 147th Infantry, Camp Shelby, Miss.
ZELLER, Robert F., 1st Lieut., Mansfield, 112th Medical Regiment, Camp Selby, Miss.

OKLAHOMA

ANDERSON, Parkey H., Major, Anadarko, 158th Field Artillery, Camp Barkeley, Texas.
BAKER, Alfred T., Captain, Durant, 180th Infantry, Fort Sill, Okla.
BAKER, Roscoe C., Major, Enid, 189th Field Artillery, Camp Barkeley.
BOLEND, Rex G., Colonel, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
BOND, Ira T., Jr., Captain, Enid, Special Troops, 45th Division.
CLOUDMAN, Harry H., Lieut. Colonel, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
DALY, John F., Captain, Pawhuska, 120th Med. Regt., Camp Barkeley.
DAVIDSON, Wallace N., Major, Cushing, 120th Medical Regiment, Camp Barkeley, Texas.
DAVIS, Thomas H., Captain, Tulsa, 120th Med. Regt., Camp Barkeley.
DEATON, Andy N., Major, Wewoka, 160th Field Artillery.
DUNNINGTON, William G., Captain, Cherokee, 189th Field Artillery, Camp Barkeley, Texas.
ENSEY, James E., Major, Altus, 120th Quartermaster Regt., Fort Sill.
FOWLER, Arthur, Jr., Captain, Sulphur, 158th Field Artillery, Camp Barkeley, Texas.
HAMMOND, James H., Capt., Tulsa, 120th Med. Regt., Camp Barkeley.
HEMPHILL, Paul H., Captain, Pawhuska, 120th Medical Regiment, Camp Barkeley, Texas.
HOLCOMBE, Roland N., Major, Muskogee, 180th Inf., Fort Sill, Okla.
HOOD, James O., Captain, Norman, 179th Infantry, Fort Sill, Okla.
KAISER, George L., Captain, Muskogee, 180th Infantry, Fort Sill, Okla.
LAWSON, Patrick H., Major, Marietta, 179th Infantry, Fort Sill, Okla.
LeHEW, Elton W., Captain, Pawnee, 179th Infantry, Fort Sill, Okla.
LINDSTROM, William C., Captain, Oklahoma City, 120th Engineers, Camp Barkeley, Texas.
McDONALD, Glen W., Captain, Ada, 120th Med. Regt., Camp Barkeley.
McKINNEY, Milam F., Captain, Oklahoma City, 120th Quartermaster Regiment, Fort Sill, Okla.
MILES, Walter H., Major, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
OGLESBEE, Vernon L., Captain, Muskogee, 180th Infantry, Fort Sill.
OHL, Charles W., 1st Lieut., Chickasha, 189th Field Artillery, Camp Barkeley, Texas.
PERRY, Daniel L., Major, Cushing, 120th Med. Regt., Camp Barkeley.
RITZHAUPT, Louis H., Major, Guthrie, State Staff.
SANGER, Fenton A., Major, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
SMITH, Lester P., Captain, Marlow, 179th Infantry, Fort Sill, Okla.
STARKEY, Wayne A., Captain, Altus, 120th Quartermaster Regiment, Fort Sill, Okla.
TACKETT, Orville H., Captain, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
TAYLOR, Lewis C., Captain, Oklahoma City, 120th Medical Regiment, Camp Barkeley, Texas.
TRACY, Gilbert W., Captain, Erick, 120th Med. Regt., Camp Barkeley.
WALTRIP, Jesse R., Captain, Yale, 158th Fld. Artillery, Camp Barkeley.
WEBSTER, William H., 1st Lieut., Ada, 120th Medical Regiment, Camp Barkeley, Texas.
WOLFE, Ira C., Captain, Muskogee, 180th Infantry, Fort Sill, Okla.

OREGON

ABBOTT, George E., 1st Lieut., Portland, 218th Field Artillery, Fort Lewis, Wash.
BAIN, Lyle M., Captain, Albany, 116th Medical Regiment, Fort Lewis.
CHAPMAN, William H., Captain, Eugene, 186th Infantry, Fort Lewis.
DAVIS, Thomas A., Major, Portland, 218th Field Artillery, Fort Lewis.
GREGSON, William E., Captain, Portland, 162d Infantry, Fort Lewis.
MILLER, Vern W., Captain, Salem, 116th Medical Regiment, Fort Lewis.
PARKS, Earl W., Captain, Portland, 162d Infantry, Fort Lewis, Wash.
RANKIN, John S., Captain, Portland, 218th Field Artillery, Fort Lewis.
ROGERS, Richard S., Captain, Junction City, 186th Inf., Fort Lewis.
ROSS, Webster K., Captain, LaGrande, 186th Inf., Fort Lewis, Wash.
SLEETER, Robert W., 1st Lieut., Medford, 186th Inf., Fort Lewis.
STANARD, Delbert C., Major, Eugene, 186th Infantry, Fort Lewis.
TUPKER, Eugene P., Captain, Salem, 116th Med. Regt., Fort Lewis.
WHITE, John BeVier, Major, Portland, 162d Infantry, Fort Lewis.
WOOD, Robert L., Major, Salem, 249th Coast Artillery.

PENNSYLVANIA

ALLEN, Robert E., Major, Mount Carmel, 190th Field Artillery, Camp Shelby, Miss.
APPEL, James Z., Capt., Lancaster, 103d Med. Regt., Indiantown Gap.
BAXTER, John G., Captain, Phoenixville, 107th Field Artillery, Indiantown Gap, Pa.

BLAND, George W., Major, Philadelphia, 111th Infantry.
BOGER, John D., Lieut. Colonel, Lebanon, 103d Medical Regiment, Indiantown Gap, Pa.
BOYSON, William A., Major, Mechanicsburg, 103d Quartermaster Regt.
BROWN, Harry, 1st Lieut., Glenmoore, 111th Infantry.
CLELAND, Charles E., Major, Kane, 112th Infantry, Indiantown Gap.
COCHRAN, Harry A. Jr., Captain, Pittsburgh, 176th Field Artillery, Fort George G. Meade, Md.
COLLEY, Arthur T., Captain, Retreat, 109th Field Artillery.
COPPES, Charles D., 1st Lieut., Philadelphia, 104th Cavalry.
DAUGHERTY, Charles B., Lieut. Colonel, Tyrone, 103d Medical Regiment, Indiantown Gap, Pa.
DEATERLY, Charles F., 1st Lieut., Quakertown, 166th Field Artillery, Camp Shelby, Miss.
DIEHL, Kenneth L., 1st Lieut., Pitsburgh, 110th Inf., Indiantown Gap.
DOANE, John H., Lieut. Colonel, Mansfield, 103d Medical Regiment, Indiantown Gap, Pa.
DONALDSON, Arthur V., Captain, Canonsburg, 103d Medical Regiment, Indiantown Gap, Pa.
DOUDS, Harry E., Captain, Beaver Falls, 103d Medical Regiment, Indiantown Gap, Pa.
DOYLE, Albert F., Captain, Johnstown, 103d Quartermaster Regiment.
DUTTENHOFER, Charles S., Lieut. Colonel, Churchtown, 103d Medical Regiment, Indiantown Gap, Pa.
EVERHART, Edgar S., Lieut. Colonel, Lemoyne, 122d Medical Squadron.
FARIES, George B., Capt., Lewisburg, 190th Fld. Artillery, Camp Shelby.
FARQUHAR, George A., Major, Monongahela, 110th Infantry, Indiantown Gap, Pa.
FEIGHTNER, Francis W., 1st Lieut., Greensburg, 110th Infantry, Indiantown Gap, Pa.
FUNKE, Alvin H., Captain, Ashley, 103d Med. Regt., Indiantown Gap.
GIBSON, William Blake, 1st Lieut., Lansdowne, 108th Field Artillery, Indiantown Gap, Pa.
HANNA, Edward A., 1st Lieut., Philadelphia, 122d Medical Squadron.
HAUCK, Samuel M., Capt., Lancaster, 103d Med. Regt., Indiantown Gap.
HAZLETT, Esten L., Captain, Canonsburg, 103d Medical Regiment, Indiantown Gap, Pa.
HENDRICKS, Charles S., Lieut. Colonel, Altoona, 103d Medical Regiment, Indiantown Gap, Pa.
HODGSON, Eugene W., 1st Lieut., Houston, 103d Medical Regiment, Indiantown Gap, Pa.
HOFFSTEIN, Louis D., 1st Lieut., Philadelphia, 111th Infantry.
HOGG, Harold K., Major, Lancaster, 103d Med. Regt., Indiantown Gap.
HOOVER, Carl H., Capt., Lancaster, 103d Med. Regt., Indiantown Gap.
HOWLAND, Harry W., Captain, Galeton, 103d Medical Regiment, Indiantown Gap, Pa.
INGOLDSBY, Eugene C., Captain, Altoona, 190th Field Artillery, Camp Shelby, Miss.
KEENEY, Paul A., Major, Harrisburg, 104th Cavalry.
KOCHENDERFER, Thomas T., 1st Lieut., Norristown, 108th Field Artillery, Indiantown Gap, Pa.
LA BELLE, Charles F., Captain, Dunmore, 109th Infantry.
LAIRD, Archibald, Captain, Wellsboro, 103d Med. Regt., Indiantown Gap.
LAME, Louis A., 1st Lieut., Canonsburg, 103d Medical Regiment, Indiantown Gap, Pa.
LARKIN, Francis L., Captain, Uniontown, 110th Inf., Indiantown Gap.
LONG, Michael R., Captain, Lawrenceville, 103d Medical Regiment, Indiantown Gap, Pa.
LYNCH, James J., 1st Lieut., Philadelphia, 108th Field Artillery, Indiantown Gap, Pa.
MC BURNEY, Harold H., Captain, Washington, 103d Medical Regiment,
MC LAREN, Harold J., Major, New Brighton, 110th Infantry, Indiantown Gap, Pa.
MEISER, Edgar W., Captain, Lancaster, 103d Medical Regiment, Indiantown Gap, Pa.
NARDONE, Anthony A., Captain, Philadelphia, Special Troops, 28th Div.
PARKER, William S., Major, Bryn Mawr, 166th Field Artillery, Camp Shelby, Miss.
PASTOR, Louis M., Lieut. Colonel, Philadelphia, 103d Engineers, Indiantown Gap, Pa.
PAVLATOS, August C., Captain, Lancaster, 103d Medical Regiment, Indiantown Gap, Pa.
POHL, William F., Captain, Butler, 112th Infantry, Indiantown Gap, Pa.
POPKY, Herman B., 1st Lieut., Wilkes-Barre, 103d Medical Regiment, Indiantown Gap, Pa.
ROSENBERG, Milton M., Major, Scranton, 109th Infantry.
RUSBRIDGE, Harold W., Captain, Lebanon, 107th Field Artillery, Indiantown Gap, Pa.
SIEGEL, John M., 1st Lieut., Butler, 112th Infantry, Indiantown Gap.
SIGMOND, Henry I., Major, Philadelphia, 103d Engin., Indiantown Gap.
SKINNER, William F., Captain, Easton, 213th Coast Artillery, Camp Pendleton, Va.
SRODES, William G., Major, Woodville, 107th Field Artillery, Indiantown Gap, Pa.
STAHR, Charles P., Colonel, Lancaster, 103d Medical Regiment, Indiantown Gap, Pa.
SWIFT, John E., Major, Scranton, 109th Infantry.
ULRICH, Samuel D., Captain, Philadelphia, 108th Field Artillery, Indiantown Gap, Pa.
WARD, Frederick W., 1st Lieut., Easton, 213th Coast Artillery, Camp Pendleton, Va.
WEEST, Harry W., Jr., Lieut. Colonel, Altoona, 110th Infantry, Indiantown Gap, Pa.
WEIGEL, John E., Captain, Pittsburgh, 176th Field Artillery, Fort Geo. G. Meade, Md.
WELCH, John G., 1st Lieut., Lancaster, 103d Medical Regiment, Indiantown Gap, Pa.
WHITE, William F., Captain, Wellsboro, 103d Medical Regiment, Indiantown Gap, Pa.

RHODE ISLAND

CRANE, George E., Captain, Providence, 243d Coast Artillery, Fort Adams, R. I.
DREW, Robert W., 1st Lieut., Warren, 103d Field Artillery, Camp Blanding, Fla.
EGAN, Thomas A., Capt., Providence, 118th Med. Regt., Camp Blanding.
GANNON, Charles H., Major, Providence, 118th Engineers.
GROVER, Morris L., Captain, Providence, 118th Medical Regiment, Camp Blanding, Fla.
HEALEY, James P., 1st Lieut., Central Falls, 118th Medical Regiment, Camp Blanding, Fla.
KEEGAN, George A., 1st Lieut., Providence, 118th Engineers.
KENT, Joseph C., Major, Pawtucket, 103d Fld. Artillery, Camp Blanding.
MARTIN, Thomas A., Captain, Providence, 103d Field Artillery, Camp Blanding, Fla.
MORAN, James B., 1st Lieut., Wallum Lake, 118th Medical Regiment, Camp Blanding, Fla.
NORMANDIN, Louis A., Jr., Lieut. Colonel, 118th Medical Regiment, Camp Blanding, Fla.
PIANKA, Wallace J., 1st Lieut., Providence, 118th Medical Regiment, Camp Blanding, Fla.
ROGELL, Harold I., Major, Providence, 243d Coast Artillery, Fort Adams, R. I.
SAYER, Edmund A., Captain, Providence, 152d Observation Squadron.
WILSON, Lloyd C., Major, Providence, State Staff.

SOUTH CAROLINA

BALL, Robert W., Captain, Columbia, 263d Coast Artillery, Camp Stewart, Ga.
BARRON, William T., Capt., Columbia, 118th Inf., Fort Jackson, S. C.
BISHOP, Walter G., Captain, Greenwood, 263d Coast Artillery, Camp Stewart, Ga.
BLALOCK, George R., Capt., Clinton, 178th Field Artillery, Fort Bragg.
BRABHAM, James C., 1st Lieut., Walterboro, 178th Field Artillery, Fort McClellan, Ala.
BUCHANAN, John C., Jr., Major, Winnsboro, 105th Quartermaster Regt.
CHAPPELL, Buford S., Capt., Columbia, 118th Inf., Fort Jackson, S. C.
COLEMAN, Marshall J., Captain, Darlington, 178th Field Artillery, Fort Bragg, N. C.
EPPS, George L., 1st Lieut., Newberry, 105th Quartermaster Regiment.
FOUCHE, James William, Captain, Columbia, 105th Quartermaster Regt.
HUTCHINSON, Manly E., Major, Columbia, 263d Coast Artillery, Fort Moultrie, S. C.
JOSEY, Allen L., Captain, Columbia, 263d Coast Artillery, Fort Moultrie.
MATTHEWS, Rudolph S., Captain, Columbia, 118th Inf., Fort Jackson.
MC GOWAN, Robert P., Major, Laurens, 178th Fld. Artil., Fort Bragg.
MILES, Louis S., Captain, Summerville, 118th Inf., Fort Jackson, S. C.
SCHAYER, Isadore, Major, Columbia, 118th Inf., Fort Jackson, S. C.

SOUTH DAKOTA

AULD, Merritt A., Captain, Yankton, 109th Quartermaster Regiment.
HANSEN, Harold F., Captain, Vermillion, 147th Field Artillery, Fort Ord, Calif.
JONES, John P., Captain, Mitchell, 109th Quartermaster Regiment.
MC CARTHY, Paul V., Major, Aberdeen, 109th Engineers, Camp Claiborne, La.
MC GONIGLE, James P., Captain, Rapid City, 109th Engineers, Camp Claiborne, La.
OWEN, Gordon S., Captain, Rapid City, 109th Engineers, Camp Claiborne, La.
SACKETT, Roy F., Major, Parker, State Staff.
WILLIAMS, Forrest E., Major, Wakonda, 147th Field Artillery, Fort Ord, Calif.

TENNESSEE

CLEMENTS, Claudius A., 1st Lieut., Whitwell, 181st Field Artillery, Camp Forrest, Tenn.
CULBERTSON, John A., 1st Lieut., Clarksville, 191st Field Artillery, Camp Forrest, Tenn.
FANCHER, James R., Captain, Chattanooga, 181st Field Artillery, Camp Forrest, Tenn.
FENN, Joseph W., Major, Nashville, State Staff.
HARRIS, Frank F., Major, Chattanooga, 181st Field Artillery, Camp Forrest, Tenn.
HELLMANN, Robert S., Capt., Jackson, 117th Inf., Fort Jackson, S. C.
HENDERSON, Raymond D., Major, Memphis, 115th Field Artillery.
HUEY, Ellis J., Captain, Memphis, 115th Field Artillery.
PATTERSON, Robert C., Jr., 1st Lieut., Nashville, 105th Observation Squadron, Fort Jackson, S. C.
SMITH, Moore J., Jr., Captain, Chattanooga, 181st Field Artillery, Camp Forrest, Tenn.
SUTHERLAND, Arthur J., Jr., Major, Nashville, 191st Field Artillery, Camp Forrest, Tenn.
TAYLOR, Alva R., Captain, Jackson, 117th Infantry, Fort Jackson, S. C.
THOMPSON, Jan R., Jr., Major, Jackson, 117th Inf., Fort Jackson.
YANCEY, Charles R., 1st Lieut., Nashville, 191st Field Artillery, Camp Forrest, Tenn.

TEXAS

ARCHER, James T., Jr., 1st Lieut., Houston, 111th Engineers.
BARTLETT, Henry L., Major, Houston, 124th Cav., Fort Brown, Texas.
BECK, John W. E. H., Lieut. Colonel, Austin, 11th Medical Regiment, Camp Bowie, Texas.
BEHRENS, Charles A., 1st Lieut., Houston, 141st Inf., Camp Bowie.
BRADY, Randle J., Captain, Houston, 124th Cavalry, Fort Brown, Tex.
BREWSTER, Clarence B., Major, Fort Worth, 111th Medical Regiment, Camp Bowie, Texas.
COLEMAN, Robert H., Major, Mineola, 144th Inf., Camp Bowie, Texas.
COOPWOOD, Joseph B., Captain, Lockhart, 141st Inf., Camp Bowie.
FARLEY, William W., 1st Lieut., Amarillo, 142d Inf., Camp Bowie.

FROST, Jack E., Major, DeKalb, 111th Medical Regiment, Camp Bowie.
GIESECKE, Carl G., Captain, San Antonio, 133d Field Artillery, Camp Bowie, Texas.
HARDY, John M., Captain, Gainesville, 111th Med. Regt., Camp Bowie.
HARGIS, William H., Sr., Major, San Antonio, 133d Field Artillery, Camp Bowie, Texas.
HARWOOD, George W., Jr., 1st Lieut., Dallas, 144th Inf., Camp Bowie.
HAWK, Hiram P., Captain, Gainesville, 111th Med. Regt., Camp Bowie.
HEFFNER, Edward A., Jr., Captain, Fort Worth, 111th Medical Regiment, Camp Bowie, Texas.
HONEA, Thomas C., Major, Cleburne, 132d Fld. Artillery, Camp Bowie.
HOPKINS, Y. Frank, Captain, Taylor, 143d Infantry.
HOWERTON, Ernest E., Captain, San Antonio, 141st Inf., Camp Bowie.
HUTCHINSON, William A., Major, Texarkana, 111th Medical Regiment, Camp Bowie, Texas.
JERABECK, John D., 1st Lieut., Houston, 124th Cav., Fort Brown, Tex.
JOHNSON, Malcolm L., 1st Lieut., Paris, 111th Medical Regiment, Camp Bowie, Texas.
LANDERS, Gardner H., Captain, Batesville, Ark., 111th Medical Regiment, Camp Bowie, Texas.
LASATER, Waldo B., Major, Mineral Wells, 112th Cavalry, Fort Clark, Texas.
LATSON, Harvey H., Major, Amarillo, 131st Field Artillery.
LETTEER, Clarence Ralph, Jr., 1st Lieut., San Antonio, Special Troops, 36th Division, Camp Bowie, Texas.
LOVING, Dan H., Major, Amarillo, 142d Infantry, Camp Bowie, Texas.
LUMPKIN, Samuel H., 1st Lieut., Amarillo, 131st Field Artillery.
MATTHEWS, John L., Captain, San Antonio, Special Troops, 36th Division, Camp Bowie, Texas.
MC GEE, Ellis B., Captain, New Boston, 111th Medical Regiment, Camp Bowie, Texas.
MEYER, Paul R., Captain, Port Arthur, 111th Engineers.
MILER, Frank P., Captain, Austin, 131st Field Artillery.
MOODY, Joe V., 1st Lieut., Dallas, 144th Infantry, Camp Bowie, Texas.
MOORE, Simm H., Major, Houston, 111th Med. Regt., Camp Bowie.
O'REILLY, John J., Colonel, Fort Worth, 111th Med. Regt., Camp Bowie.
PARMLEY, Van S., 1st Lieut., Dallas, 142d Inf., Camp Bowie, Texas.
PATE, Joe J., Captain, Paducah, 142d Infantry, Camp Bowie, Texas.
PEDIGO, Paul C., Captain, Strawn, 112th Cavalry, Fort Clark, Texas.
PICKENS, Jay W., Captain, Cleburne, 132d Field Artillery, Camp Bowie.
PINNELL, Robert, 1st Lieut., Wichita Falls, 111th Medical Regiment, Camp Bowie, Texas.
POPE, Fielding M., Captain, West, 143d Infantry.
PORTER, John T., Captain, Texarkana, Ark., 111th Medical Regiment, Camp Bowie, Texas.
PRIMER, Benjamin M., Major, Austin, 111th Med. Regt., Camp Bowie.
RATHGEB, Irving R. Jr., 1st Lieut., Texarkana, Ark., 111th Medical Regiment, Camp Bowie, Texas.
REEMTSMA, William, Captain, New Braunfels, 133d Field Artillery, Camp Bowie, Texas.
ROSS, Abner A., Major, Brownwood, 141st Infantry, Camp Bowie, Tex.
SCHMITT, Herbert S., Captain, Malakoff, 144th Inf., Camp Bowie, Tex.
SHEFFIELD, Lloyd D., Captain, Dallas, 144th Inf., Camp Bowie, Texas.
SLOAN, Roy C., Captain, Terrell, 144th Infantry, Camp Bowie, Texas.
STAHL, Louis J., Major, Gonzales, 111th Engineers.
WATT, Terrence N., 1st Lieut., Austin, 111th Quartermaster Regiment.
WIESNER, William A., 1st Lieut., San Antonio, 133d Field Artillery, Camp Bowie, Texas.
WILLIAMS, Charles R., Captain, Mineral Wells, 112th Cavalry, Fort Clark, Texas.
WILLIAMS, William E., Jr., Major, Austin, 111th Quartermaster Regt.
YATER, Tolbert F., Captain, Cleburne, 132d Fld. Artillery, Camp Bowie.

UTAH

BELNAP, Howard K., Captain, Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
CLARK, John H., Captain, Vernal, 145th Field Artillery, Camp San Luis Obispo, Calif.
CONROY, Francis R., Major, Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
GOTTFREDSON, David B., Major, Richfield, 115th Engineers, Camp San Luis Obispo, Calif.
JOHNS, Chester T., 1st Lieut., Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
MAYO, John F., 1st Lieut., Salt Lake City, 222d Field Artillery, Camp San Luis Obispo, Calif.
PRICE, Joseph L., 1st Lieut., Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
SHARP, Harlow B., Captain, Salt Lake City, 222d Field Artillery, Camp San Luis Obispo, Calif.
SHARP, John F., Major, Salt Lake City, State Staff.
SNOW, Virgil G., 1st Lieut., Cedar City, 222d Field Artillery, Camp San Luis Obispo, Calif.
WHITING, Quinn A., 1st Lieut., East Ely, Nev., 222d Field Artillery, Camp San Luis Obispo, Calif.

VERMONT

BOGLE, Bascom, Captain, Northfield, 172d Infantry, Camp Blanding, Fla.
COOTEY, Sherwin A., Lieut. Colonel, Rutland, 118th Medical Regiment.
DUNSMORE, Murray K., Major, St. Albans, 118th Quartermaster Regiment, Camp Blanding, Fla.
FARMER, Henry M., Captain, Burlington, 172d Inf., Camp Blanding.
FARMER, Howard J., 1st Lieut., St. Johnsbury, 172d Infantry, Camp Blanding, Fla.
KENT, Fred S., Major, Burlington, 172d Infantry, Camp Blanding, Fla.
SHELC, Joseph P., 1st Lieut., Bellows Falls, 172d Inf., Camp Blanding.
SIMONDS, John R., 1st Lieut., Woonsocket, R. I., 118th Quartermaster Regiment, Camp Blanding, Fla.
SOUTHWORTH, John D., Captain, Rutland, 118th Medical Regiment.

VIRGINIA

ALLISON, Robley C., 1st Lieut., Petersburg, 176th Infantry.
BARROW, Frederick P., II, Major, Portsmouth, 111th Field Artillery, Fort George G. Meade, Md.
BOWEN, Richard A., II, Major, Richmond, 176th Infantry.
BROWN, Hugh B., Jr., 1st Lieut., Draper, 116th Inf., Fort Meade, Md.
BUCKLEY, Millard R., Captain, Richmond, 111th Field Artillery, Fort George G. Meade, Md.
DAVIS, Charles F., Jr., Captain, Roanoke, 246th Coast Artillery, Fort Story, Va.
HURT, Holcombe H., Captain, Virginia Beach, 246th Coast Artillery, Fort Story, Va.
HURT, Ira H., Major, Roanoke, 246th Coast Artillery, Fort Story, Va.
JOHNSON, Garnett W., Captain, Danville, 104th Medical Regiment.
LUSH, Lewis Chasteen, Major, Richmond, 176th Infantry.
POWELL, Rupert W., 1st Lieut., Roanoke, 116th Infantry, Fort Meade.
RISHER, John C., 1st Lieut., Lynchburg, 246th Coast Artillery, Fort Story, Va.
RUFFIN, Herbert G., 1st Lieut., Arvonia, 104th Medical Regiment.
SOUTHWARD, Wilbur R., Jr., Major, Richmond, 104th Medical Regt.
TRICE, Ernest T., Lieut. Colonel, Richmond, State Staff.
WEAVER, Oswald M., 1st Lieut., Colony, 116th Infantry, Fort Meade.
WHITE, Harry F., Major, Fishersville, 116th Infantry, Fort Meade, Md.

WASHINGTON

BAUGH, Floyd W., Major, Burlington, 116th Med. Regt., Fort Lewis.
BEELER, George W., Colonel, Seattle, 116th Medical Regt., Fort Lewis.
BENJAMIN, Mac B., Captain, Tacoma, 103d Anti-Tank Battalion, Fort Lewis, Wash.
BENNER, Stanley R., Captain, Yakima, 161st Infantry, Fort Lewis.
BINGHAM, Harvey D., Capt., Seattle, 205th Coast Artillery, Fort Lewis.
BURNS, Francis J., Major, Spokane, 161st Infantry, Fort Lewis, Wash.
CARTER, Ronald P., Captain, Seattle, 116th Medical Regt., Fort Lewis.
COLE, Harold C., 1st Lieut., Seattle, 116th Medical Regt., Fort Lewis.
FRANZ, Francis W., Captain, Seattle, 116th Medical Regiment, Fort Lewis, Wash.
HALVORSEN, Clifford, Captain, Fort Steilacoom, 248th Coast Artillery, Fort Worden, Wash.
HUNTER, Maxwell R., Captain, Olympia, 205th Coast Artillery, Fort Lewis, Wash.
JOHNSON, Willis B., Captain, Everett, 161st Infantry, Fort Lewis.
JONES, Scott S., Major, Tacoma, 248th Coast Artillery, Fort Worden.
KAVENEY, Irving E., 1st Lieut., Port Angeles, 116th Medical Regiment, Fort Lewis, Wash.
KLING, George W., Captain, Centralia, Special Troops, 41st Division, Fort Lewis, Wash.
KYLE, Philip Collins, Major, Tacoma, 116th Quartermaster Regiment.
LAIGE, Raymond, Captain, Seattle, 146th Field Artillery.
LILLIBRIDGE, Harold D., Major, Olympia, 205th Coast Artillery, Fort Lewis, Wash.
LINK Richard B., Captain, Tacoma, 248th Coast Artillery, Fort Worden.
MILLER, Richard R., Captain, Seattle, 116th Med. Regt., Fort Lewis.
OSBORNE, Lester F., 1st Lieut., Tacoma, 116th Med. Regt., Fort Lewis.
PARMELEE, Ernest B., Captain, Seattle, 116th Quartermaster Regt.
TSAPRALIS, Paul Constantine, 1st Lieut., Seattle, 116th Medical Regiment, Fort Lewis, Wash.
WHYTE, Kenneth G., Major, Seattle, 116th Med. Regt., Fort Lewis.

WEST VIRGINIA

BRICK, John P., 1st Lieut., Charleston, 150th Inf., Camp Shelby, Miss.
CARNEY, Harry B. A., Captain, Charleston, 150th Inf., Camp Shelby.
COPLIN, Robert W., 1st Lieut., Huntington, 150th Inf., Camp Shelby.
EVANS, George T., 1st Lieut., Fairmont, 201st Infantry, Fort Benjamin Harrison, Ind.
FORD, Joseph C., Captain, Huntington, 150th Inf., Camp Shelby, Miss.
HARMAN, Robert D., Major, Kingwood, 201st Inf., Fort Harrison, Ind.
LAMBERT, Luther R., Major, Fairmont, State Staff.
POST, Donald M., Captain, Morgantown, 201st Inf., Fort Harrison, Ind.
VAN METRE, Robert S., Major, Huntington, 150th Inf., Camp Shelby.

WISCONSIN

BECKER, Reinhard, Major, Milwaukee, 135th Med. Regt., Camp Shelby.
BECKER, Walter T., Captain, Cederburg, 135th Medical Regiment, Camp Shelby, Miss.
BLECKWENN, William J., Colonel, Madison, 135th Medical Regiment, Camp Shelby, Miss.
BOXER, Leo M., Captain, Milwaukee, 135th Med. Regt., Camp Shelby.
BROOKE, James W., 1st Lieut., Madison, 135th Medical Regiment, Camp Shelby, Miss.
CHRISTIANSEN, Wilmer H., Captain, Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
CHRISTIANSON, Herbert B., 1st Lieut., Superior, 128th Infantry, Camp Livingston, La.
EAGLEBURGER, Leon S., Major, Waupun, 135th Medical Regiment, Camp Shelby, Miss.
FEIN, Bernard T., 1st Lieut., Milwaukee, 127th Inf., Camp Beauregard.
FERRIS, James W., Capt., Wausau, 107th Quartermaster Regiment, Camp Beauregard, La.
GALLOGLY, John A., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
GRAB, John A., Major, Milwaukee, 135th Medical Regt., Camp Shelby.
HATHAWAY, George J., Major, Superior, 128th Inf., Camp Livingston.
HAUKOHL, Robert S., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
HEIDEN, Harry H., Major, Sheboygan, 107th Quartermaster Regiment, Camp Beauregard, La.
HOLLENBECK, Stanley W., Major, Milwaukee, 126th Field Artillery.

HOLMES, John F., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
KENDALL, Earl T., Captain, Racine, 135th Med. Regt., Camp Shelby.
LEVEROOS, Edward H., 1st Lieut., Superior, 128th Infantry, Camp Livingston, La.
MC CABE, John O., Capt., Milwaukee, 135th Med. Regt., Camp Shelby.
MC MAHON, Raymond A., 1st Lieut., Madison, 135th Medical Regiment, Camp Shelby, Miss.
MILLER, James E., Captain, Madison, 135th Med. Regt., Camp Shelby.
MORAN, Clement J., Captain, La Crosse, 120th Field Artillery, Camp Beauregard, La.
MUENZNER, Richard J., Jr., 1st Lieut., Milwaukee, 121st Field Artillery, Camp Beauregard, La.
MUSSER, Marc J., Jr., Major, Madison, 135th Med. Regt., Camp Shelby.
NEBEL, Harold, Major, Milwaukee, 121st Field Artillery, Camp Beauregard, La.
NOWACK, Louis W., Captain, Watertown, 127th Inf., Camp Beauregard.
PETERSON, John R., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
PETERSON, Leo W., Lieut. Colonel, Sun Prairie, 135th Medical Regiment, Camp Shelby, Miss.
POHLE, Frederick J., Captain, Madison, 135th Med. Regt., Camp Shelby.
SCHRAMEL, Anton J., Captain, Milwaukee, 120th Field Artillery, Camp Beauregard, La.
SCHWARZE, Cyril A., 1st Lieut., Watertown, 135th Medical Regiment, Camp Shelby, Miss.
SHIELDS, Herbert B., 1st Lieut., Milwaukee, 127th Infantry, Camp Beauregard, La.
THANOS, John N., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
VETTER, Edward W., Capt., Randolph, 135th Med. Regt., Camp Shelby.
WEISSMILLER, Lester L., Captain, Madison, 135th Medical Regiment, Camp Shelby, Miss.
WILKINSON, Philip M., Captain, Oconomowoc, 135th Medical Regiment, Camp Shelby, Miss.
ZINTEK, Sylvester S., Major, Milwaukee, 127th Infantry, Camp Beauregard, La.

WYOMING

PENNOYER, Willard H., Captain, Cheyenne, 115th Cavalry, Fort Lewis.
RAE, Harold B., Major, Torrington, 115th Cavalry, Fort Lewis, Wash.

ALASKA

TOOHEY, Clarence J., Captain, Anchorage, State Staff.
VOLLERT, Edward F., Captain, Juneau, 297th Infantry.

HAWAII

AVAKIAN, Edward V., Captain, Pepeekeo, 299th Infantry, Schofield Barracks, Hawaii.
COCKETT, Patrick M., Captain, Wailuku, 299th Infantry, Schofield Barracks, Hawaii.
KONG, Raymond F., Captain, Honolulu, 298th Infantry, Schofield Barracks, Hawaii.
PANG, David L., Capt., Honolulu, 298th Inf., Schofield Barracks, Hawaii.
PHILLIPS, Clyde L., 1st Lieut., Hilo, 299th Inf., Schofield Barracks.
TESSMER, Carl F., Capt., Honolulu, 298th Inf., Schofield Barracks.
TONG, Fook H., Captain, Honolulu, 299th Infantry., Schofield Barracks.

PUERTO RICO

BUSO-CARRASQUILLO, Roberto, Captain, San Juan, 296th Infantry, Camp Tortuguero, P. R.
de DIEGO, Manuel, Captain, Ponce, 162d Field Artillery.
de la VEGA, Luis B., Major, Rio Piedras, State Staff.
GALLARDO, Jose A., 1st Lieut., San Juan, 295th Infantry.
GUZMAN, Manuel, Jr., Captain, Mayaguez, 296th Infantry, Camp Tortuguero, P. R.
JULIA, Carlos M., Captain, San Juan, 295th Infantry.
PASSALACQUA, Luis A., Captain, Ponce, 296th Infantry, Camp Tortuguero, P. R.
RAMIREZ, Ramon E., Captain, Mayaguez, 296th Infantry, Camp Tortuguero, P. R.
SILVA, Euripides, Major, San Juan, 296th Inf., Camp Tortuguero, P. R.

NONPERISHABLE FOOD FOR THE ARMY

The War Department has announced that continental United States will be divided into nine areas to facilitate the purchase, storage and distribution of nonperishable food for the Army. The nine quartermaster or general depots will be located at Boston, Chicago, San Francisco, Atlanta, Ga., Charlotte, N. C., New Orleans, San Antonio, Texas, Kansas City, Mo., and Jersey City, N. J., each depot to serve for this purpose various surrounding states. However, in the procurement of certain nonperishable foods, only three of the nine depots will be used. The Chicago depot will procure canned meats and will handle also canned vegetables, milk, dried eggs and wheat flour, except for the area served by the San Francisco depot, which will procure its own wheat flour. The San Francisco depot in addition will procure canned apricots, cherries, peaches, pears, pineapples and prunes; also canned asparagus and canned salmon. The Jersey City depot will procure canned apples, apple sauce, apple butter, coffee, canned grape fruit, jam, preserves, sugar, syrup, salt, tea and flavoring extracts. Among the items to be procured by each of the regional depots except Charlotte and Kansas City are canned blackberries, blueberries, loganberries, raspberries, canned sour cherries, canned cabbage, carrots, rice, sugar, spices, black pepper, vinegar, canned sardines, canned tuna fish, ground cinnamon, cocoa, gelatin and desserts, powders and peanut butter. Such items for the Charlotte area will be purchased by the Jersey City depot, and Kansas City will be furnished through the Chicago depot. Normally all of these items, with the exception of flour and green bean coffee, will be purchased f. o. b. at the army post, camp or station where they are to be used. If the carload shipment cannot be made, deliveries will be made to the regional depot, where they will be pooled with other items, when practicable, to make up carload lots.

YELLOW FEVER VACCINATIONS COMPLETED IN PUERTO RICO

Inoculation of the entire command at Puerto Rico against yellow fever has been completed without a single case of "unusual reaction," the War Department announced July 2. It was the largest mass yellow fever inoculation ever attempted in the history of the United States Army.

Decision to adopt this method of protecting troops in tropical regions against yellow fever authorized earlier this year grew out of the discovery by medical science of a jungle type of the fever that has its reservoir of virus in wild life as contrasted with malaria which is transmitted only between human beings. It cannot be stamped out and immunization was adopted in order to protect soldiers against possible infection in tropical regions.

All military personnel stationed at posts in the tropical regions of the Western Hemisphere are being inoculated against yellow fever. Troops ordered to duty in these regions will be vaccinated before leaving the states.

The yellow fever vaccine used by the Army consists of a special strain of yellow fever virus that has been attenuated through prolonged cultivation in tissue cultures by the Rockefeller Institute for Medical Research, New York. Extreme care is necessary in the packing and shipping of the vaccine, since it must be carried in containers in which the temperature does not exceed 39.2 F.

INDUSTRIAL HYGIENE IN DEFENSE INDUSTRIES

Approximately $250,000 is to be made available to the Division of Industrial Hygiene of the National Institute of Health for the 1942 fiscal year in order to continue the industrial hygiene activities in defense industries which were initiated on March 1 of this year. This amount, plus the regular appropriation of the division, will bring the entire budget for industrial hygiene in the Public Health Service close to $500,000. At present there are 106 persons engaged in this work, of whom 17 are physicians, 16 are engineers, 32 are chemists, toxicologists and physicists, and 41 are subprofessional and clerical workers. There are now 32 professional persons engaged directly in field activities in defense industries. This group consists of 12 physicians, 15 engineers, 4 chemists and 1 nurse. It is planned to increase this mobile field group in order to satisfy the demands for personnel in the states.

CANADIAN DOCTORS FOR BRITAIN

Arrangements have been completed between the British Red Cross and the Canadian Red Cross to send Canadian physicians to Britain. The project is comparable to the one which is sending American physicians to aid Britain. Fifty Canadian physicians are now ready to sail and more will follow, depending on transport facilities.

FROST, Jack E., Major, DeKalb, 111th Medical Regiment, Camp Bowie.
GIESECKE, Carl G., Captain, San Antonio, 133d Field Artillery, Camp Bowie, Texas.
HARDY, John M., Captain, Gainesville, 111th Med. Regt., Camp Bowie.
HARGIS, William H., Sr., Major, San Antonio, 133d Field Artillery, Camp Bowie, Texas.
HARWOOD, George W., Jr., 1st Lieut., Dallas, 144th Inf., Camp Bowie.
HAWK, Hiram P., Captain, Gainesville, 111th Med. Regt., Camp Bowie.
HEFFNER, Edward A., Jr., Captain, Fort Worth, 111th Medical Regiment, Camp Bowie, Texas.
HONEA, Thomas C., Major, Cleburne, 132d Fld. Artillery, Camp Bowie.
HOPKINS, Y. Frank, Captain, Taylor, 143d Infantry.
HOWERTON, Ernest E., Captain, San Antonio, 141st Inf., Camp Bowie.
HUTCHINSON, William A., Major, Texarkana, 111th Medical Regiment, Camp Bowie, Texas.
JERABECK, John D., 1st Lieut., Houston, 124th Cav., Fort Brown, Tex.
JOHNSON, Malcolm L., 1st Lieut., Paris, 111th Medical Regiment, Camp Bowie, Texas.
LANDERS, Gardner H., Captain, Batesville, Ark., 111th Medical Regiment, Camp Bowie, Texas.
LASATER, Waldo B., Major, Mineral Wells, 112th Cavalry, Fort Clark, Texas.
LATSON, Harvey H., Major, Amarillo, 131st Field Artillery.
LETTEER, Clarence Ralph, Jr., 1st Lieut., San Antonio, Special Troops, 36th Division, Camp Bowie, Texas.
LOVING, Dan H., Major, Amarillo, 142d Infantry, Camp Bowie, Texas.
LUMPKIN, Samuel H., 1st Lieut., Amarillo, 131st Field Artillery.
MATTHEWS, John L., Captain, San Antonio, Special Troops, 36th Division, Camp Bowie, Texas.
MC GEE, Ellis B., Captain, New Boston, 111th Medical Regiment, Camp Bowie, Texas.
MEYER, Paul R., Captain, Port Arthur, 111th Engineers.
MILLER, Frank P., Captain, Austin, 131st Field Artillery.
MOODY, Joe V., 1st Lieut., Dallas, 144th Infantry, Camp Bowie, Texas.
MOORE, Simm H., Major, Houston, 111th Med. Regt., Camp Bowie.
O'REILLY, John J., Colonel, Fort Worth, 111th Med. Regt., Camp Bowie.
PARMLEY, Van S., 1st Lieut., Dallas, 142d Inf., Camp Bowie, Texas.
PATE, Joe J., Captain, Paducah, 142d Infantry, Camp Bowie, Texas.
PEDIGO, Paul C., Captain, Strawn, 112th Cavalry, Fort Clark, Texas.
PICKENS, Jay W., Captain, Cleburne, 132d Field Artillery, Camp Bowie.
PINNELL, Robert, 1st Lieut., Wichita Falls, 111th Medical Regiment, Camp Bowie, Texas.
POPE, Fielding M., Captain, West, 143d Infantry.
PORTER, John T., Captain, Texarkana, Ark., 111th Medical Regiment, Camp Bowie, Texas.
PRIMER, Benjamin M., Major, Austin, 111th Med. Regt., Camp Bowie.
RATHGEB, Irving R. Jr., 1st Lieut., Texarkana, Ark., 111th Medical Regiment, Camp Bowie, Texas.
REEMTSMA, William, Captain, New Braunfels, 133d Field Artillery, Camp Bowie, Texas.
ROSS, Abner A., Major, Brownwood, 141st Infantry, Camp Bowie, Tex.
SCHMITT, Herbert S., Captain, Malakoff, 144th Inf., Camp Bowie, Tex.
SHEFFIELD, Lloyd B., Captain, Dallas, 144th Inf., Camp Bowie, Texas.
SLOAN, Roy C., Captain, Terrell, 144th Infantry, Camp Bowie, Texas.
STAHL, Louis J., Major, Gonzales, 111th Engineers.
WATT, Terrence N., 1st Lieut., Austin, 111th Quartermaster Regiment.
WIESNER, William A., 1st Lieut., San Antonio, 133d Field Artillery, Camp Bowie, Texas.
WILLIAMS, Charles R., Captain, Mineral Wells, 112th Cavalry, Fort Clark, Texas.
WILLIAMS, William E., Jr., Major, Austin, 111th Quartermaster Regt.
YATER, Tolbert F., Captain, Cleburne, 132d Fld. Artillery, Camp Bowie.

UTAH

BELNAP, Howard K., Captain, Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
CLARK, John H., Captain, Vernal, 145th Field Artillery, Camp San Luis Obispo, Calif.
CONROY, Francis R., Major, Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
GOTTFREDSON, David B., Major, Richfield, 115th Engineers, Camp San Luis Obispo, Calif.
JOHNS, Chester T., 1st Lieut., Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
MAYO, John F., 1st Lieut., Salt Lake City, 222d Field Artillery, Camp San Luis Obispo, Calif.
PRICE, Joseph L., 1st Lieut., Ogden, 115th Medical Regiment, Camp San Luis Obispo, Calif.
SHARP, Harlow B., Captain, Salt Lake City, 222d Field Artillery, Camp San Luis Obispo, Calif.
SHARP, John F., Major, Salt Lake City, State Staff.
SNOW, Virgil C., 1st Lieut., Cedar City, 222d Field Artillery, Camp San Luis Obispo, Calif.
WHITING, Quinn A., 1st Lieut., East Ely, Nev., 222d Field Artillery, Camp San Luis Obispo, Calif.

VERMONT

BOGLE, Bascom, Captain, Northfield, 172d Infantry, Camp Blanding, Fla.
COOTEY, Sherwin A., Lieut. Colonel, Rutland, 118th Medical Regiment.
DUNSMORE, Murray K., Major, St. Albans, 118th Quartermaster Regiment, Camp Blanding, Fla.
FARMER, Henry M., Captain, Burlington, 172d Inf., Camp Blanding.
FARMER, Howard J., 1st Lieut., St. Johnsbury, 172d Infantry, Camp Blanding, Fla.
KENT, Fred S., Major, Burlington, 172d Infantry, Camp Blanding, Fla.
SHELC, Joseph P., 1st Lieut., Bellows Falls, 172d Inf., Camp Blanding.
SIMONDS, John R., 1st Lieut., Woonsocket, R. I., 118th Quartermaster Regiment, Camp Blanding, Fla.
SOUTHWORTH, John D., Captain, Rutland, 118th Medical Regiment.

VIRGINIA

ALLISON, Robley C., 1st Lieut., Petersburg, 176th Infantry.
BARROW, Frederick P., II, Major, Portsmouth, 111th Field Artillery, Fort George G. Meade, Md.
BOWEN, Richard A., II, Major, Richmond, 176th Infantry.
BROWN, Hugh B., Jr., 1st Lieut., Draper, 116th Inf., Fort Meade, Md.
BUCKLEY, Millard R., Captain, Richmond, 111th Field Artillery, Fort George G. Meade, Md.
DAVIS, Charles F., Jr., Captain, Roanoke, 246th Coast Artillery, Fort Story, Va.
HURT, Holcombe H., Captain, Virginia Beach, 246th Coast Artillery, Fort Story, Va.
HURT, Ira H., Major, Roanoke, 246th Coast Artillery, Fort Story, Va.
JOHNSON, Garnett W., Captain, Danville, 104th Medical Regiment.
LUSH, Lewis Chasteen, Major, Richmond, 176th Infantry.
POWELL, Rupert W., 1st Lieut., Roanoke, 116th Infantry, Fort Meade.
RISHER, John C., 1st Lieut., Lynchburg, 246th Coast Artillery, Fort Story, Va.
RUFFIN, Herbert G., 1st Lieut., Arvonia, 104th Medical Regiment.
SOUTHWARD, Wilbur R., Jr., Major, Richmond, 104th Medical Regt.
TRICE, Ernest T., Lieut. Colonel, Richmond, State Staff.
WEAVER, Oswald M., 1st Lieut., Colony, 116th Infantry, Fort Meade.
WHITE, Harry F., Major, Fishersville, 116th Infantry, Fort Meade, Md.

WASHINGTON

BAUGH, Floyd W., Major, Burlington, 116th Med. Regt., Fort Lewis.
BEELER, George W., Colonel, Seattle, 116th Medical Regt., Fort Lewis.
BENJAMIN, Mac B., Captain, Tacoma, 103d Anti-Tank Battalion, Fort Lewis, Wash.
BENNER, Stanley R., Captain, Yakima, 161st Infantry, Fort Lewis.
BINGHAM, Harvey D., Capt., Seattle, 205th Coast Artillery, Fort Lewis.
BURNS, Francis J., Major, Spokane, 161st Infantry, Fort Lewis, Wash.
CARTER, Ronald P., Captain, Seattle, 116th Medical Regt., Fort Lewis.
COLE, Harold C., 1st Lieut., Seattle, 116th Medical Regt., Fort Lewis.
FRANZ, Francis W., Captain, Seattle, 116th Medical Regiment, Fort Lewis, Wash.
HALVORSEN, Clifford, Captain, Fort Steilacoom, 248th Coast Artillery, Fort Worden, Wash.
HUNTER, Maxwell R., Captain, Olympia, 205th Coast Artillery, Fort Lewis, Wash.
JOHNSON, Willis B., Captain, Everett, 161st Infantry, Fort Lewis.
JONES, Scott S., Major, Tacoma, 248th Coast Artillery, Fort Worden.
KAVENEY, Irving E., 1st Lieut., Port Angeles, 116th Medical Regiment, Fort Lewis, Wash.
KLING, George W., Captain, Centralia, Special Troops, 41st Division, Fort Lewis, Wash.
KYLE, Philip Collins, Major, Tacoma, 116th Quartermaster Regiment.
LAIGE, Raymond, Captain, Seattle, 146th Field Artillery.
LILLIBRIDGE, Harold D., Major, Olympia, 205th Coast Artillery, Fort Lewis, Wash.
LINK Richard B., Captain, Tacoma, 248th Coast Artillery, Fort Worden.
MILLER, Richard R., Captain, Seattle, 116th Med. Regt., Fort Lewis.
OSBORNE, Lester F., 1st Lieut., Tacoma, 116th Med. Regt., Fort Lewis.
PARMELEE, Ernest B., Captain, Seattle, 116th Quartermaster Regt.
TSAPRALIS, Paul Constantine, 1st Lieut., Seattle, 116th Medical Regiment, Fort Lewis, Wash.
WHYTE, Kenneth G., Major, Seattle, 116th Med. Regt., Fort Lewis.

WEST VIRGINIA

BRICK, John P., 1st Lieut., Charleston, 150th Inf., Camp Shelby, Miss.
CARNEY, Harry B. A., Captain, Charleston, 150th Inf., Camp Shelby.
COPLIN, Robert W., 1st Lieut., Huntington, 150th Inf., Camp Shelby.
EVANS, George T., 1st Lieut., Fairmont, 201st Infantry, Fort Benjamin Harrison, Ind.
FORD, Joseph C., Captain, Huntington, 150th Inf., Camp Shelby, Miss.
HARMAN, Robert D., Major, Kingwood, 201st Inf., Fort Harrison, Ind.
LAMBERT, Luther R., Major, Fairmont, State Staff.
POST, Donald M., Captain, Morgantown, 201st Inf., Fort Harrison, Ind.
VAN METRE, Robert S., Major, Huntington, 150th Inf., Camp Shelby.

WISCONSIN

BECKER, Reinhard, Major, Milwaukee, 135th Med. Regt., Camp Shelby.
BECKER, Walter T., Captain, Cederburg, 135th Medical Regiment, Camp Shelby, Miss.
BLECKWENN, William J., Colonel, Madison, 135th Medical Regiment, Camp Shelby, Miss.
BOXER, Leo M., Captain, Milwaukee, 135th Med. Regt., Camp Shelby.
BROOKE, James W., 1st Lieut., Madison, 135th Medical Regiment, Camp Shelby, Miss.
CHRISTIANSEN, Wilmer H., Captain, Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
CHRISTIANSON, Herbert B., 1st Lieut., Superior, 128th Infantry, Camp Livingston, La.
EAGLEBURGER, Leon S., Major, Waupun, 135th Medical Regiment, Camp Shelby, Miss.
FEIN, Bernard T., 1st Lieut., Milwaukee, 127th Inf., Camp Beauregard.
FERRIS, James W., Capt., Wausau, 107th Quartermaster Regiment, Camp Beauregard, La.
GALLOGLY, John A., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
GRAB, John A., Major, Milwaukee, 135th Medical Regt., Camp Shelby.
HATHAWAY, George J., Major, Superior, 128th Inf., Camp Livingston.
HAUKOHL, Robert S., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
HEIDEN, Harry H., Major, Sheboygan, 107th Quartermaster Regiment, Camp Beauregard, La.
HOLLENBECK, Stanley W., Major, Milwaukee, 126th Field Artillery.

HOLMES, John F., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
KENDALL, Earl T., Captain, Racine, 135th Med. Regt., Camp Shelby.
LEVEROOS, Edward H., 1st Lieut., Superior, 128th Infantry, Camp Livingston, La.
MC CABE, John O., Capt., Milwaukee, 135th Med. Regt., Camp Shelby.
MC MAHON, Raymond A., 1st Lieut., Madison, 135th Medical Regiment, Camp Shelby, Miss.
MILLER, James E., Captain, Madison, 135th Med. Regt., Camp Shelby.
MORAN, Clement J., Captain, La Crosse, 120th Field Artillery, Camp Beauregard, La.
MUENZNER, Richard J., Jr., 1st Lieut., Milwaukee, 121st Field Artillery, Camp Beauregard, La.
MUSSER, Marc J., Jr., Major, Madison, 135th Med. Regt., Camp Shelby.
NEBEL, Harold, Major, Milwaukee, 121st Field Artillery, Camp Beauregard, La.
NOWACK, Louis W., Captain, Watertown, 127th Inf., Camp Beauregard.
PETERSON, John R., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
PETERSON, Leo W., Lieut. Colonel, Sun Prairie, 135th Medical Regiment, Camp Shelby, Miss.
POHLE, Frederick J., Captain, Madison, 135th Med. Regt., Camp Shelby.
SCHRAMEL, Anton J., Captain, Milwaukee, 120th Field Artillery, Camp Beauregard, La.
SCHWARZE, Cyril A., 1st Lieut., Watertown, 135th Medical Regiment, Camp Shelby, Miss.
SHIELDS, Herbert B., 1st Lieut., Milwaukee, 127th Infantry, Camp Beauregard, La.
THANOS, John N., 1st Lieut., Milwaukee, 135th Medical Regiment, Camp Shelby, Miss.
VETTER, Edward W., Capt., Randolph, 135th Med. Regt., Camp Shelby.
WEISSMILLER, Lester L., Captain, Madison, 135th Medical Regiment, Camp Shelby, Miss.
WILKINSON, Philip M., Captain, Oconomowoc, 135th Medical Regiment, Camp Shelby, Miss.
ZINTEK, Sylvester S., Major, Milwaukee, 127th Infantry, Camp Beauregard, La.

WYOMING

PENNOYER, Willard H., Captain, Cheyenne, 115th Cavalry, Fort Lewis.
RAE, Harold B., Major, Torrington, 115th Cavalry, Fort Lewis, Wash.

ALASKA

TOOHEY, Clarence J., Captain, Anchorage, State Staff.
VOLLERT, Edward F., Captain, Juneau, 297th Infantry.

HAWAII

AVAKIAN, Edward V., Captain, Pepeekeo, 299th Infantry, Schofield Barracks, Hawaii.
COCKETT, Patrick M., Captain, Wailuku, 299th Infantry, Schofield Barracks, Hawaii.
KONG, Raymond F., Captain, Honolulu, 298th Infantry, Schofield Barracks, Hawaii.
PANG, David L., Capt., Honolulu, 298th Inf., Schofield Barracks, Hawaii.
PHILLIPS, Clyde L., 1st Lieut., Hilo, 299th Inf., Schofield Barracks.
TESSMER, Carl F., Capt., Honolulu, 298th Inf., Schofield Barracks.
TONG, Fook H., Captain, Honolulu, 299th Infantry., Schofield Barracks.

PUERTO RICO

BUSO-CARRASQUILLO, Roberto, Captain, San Juan, 296th Infantry, Camp Tortuguero, P. R.
de DIEGO, Manuel, Captain, Ponce, 162d Field Artillery.
de la VEGA, Luis B., Major, Rio Piedras, State Staff.
GALLARDO, Jose A., 1st Lieut., San Juan, 295th Infantry.
GUZMAN, Manuel, Jr., Captain, Mayaguez, 296th Infantry, Camp Tortuguero, P. R.
JULIA, Carlos M., Captain, San Juan, 295th Infantry.
PASSALACQUA, Luis A., Captain, Ponce, 296th Infantry, Camp Tortuguero, P. R.
RAMIREZ, Ramon E., Captain, Mayaguez, 296th Infantry, Camp Tortuguero, P. R.
SILVA, Euripides, Major, San Juan, 296th Inf., Camp Tortuguero, P. R.

NONPERISHABLE FOOD FOR THE ARMY

The War Department has announced that continental United States will be divided into nine areas to facilitate the purchase, storage and distribution of nonperishable food for the Army. The nine quartermaster or general depots will be located at Boston, Chicago, San Francisco, Atlanta, Ga., Charlotte, N. C., New Orleans, San Antonio, Texas, Kansas City, Mo., and Jersey City, N. J., each depot to serve for this purpose various surrounding states. However, in the procurement of certain nonperishable foods, only three of the nine depots will be used. The Chicago depot will procure canned meats and will handle also canned vegetables, milk, dried eggs and wheat flour, except for the area served by the San Francisco depot, which will procure its own wheat flour. The San Francisco depot in addition will procure canned apricots, cherries, peaches, pears, pineapples and prunes; also canned asparagus and canned salmon. The Jersey City depot will procure canned apples, apple sauce, apple butter, coffee, canned grape fruit, jam, preserves, sugar, syrup, salt, tea and flavoring extracts. Among the items to be procured by each of the regional depots except Charlotte and Kansas City are canned blackberries, blueberries, loganberries, raspberries, canned sour cherries, canned cabbage, carrots, rice, sugar, spices, black pepper, vinegar, canned sardines, canned tuna fish, ground cinnamon, cocoa, gelatin and desserts, powders and peanut butter. Such items for the Charlotte area will be purchased by the Jersey City depot, and Kansas City will be furnished through the Chicago depot. Normally all of these items, with the exception of flour and green bean coffee, will be purchased f. o. b. at the army post, camp or station where they are to be used. If the carload shipment cannot be made, deliveries will be made to the regional depot, where they will be pooled with other items, when practicable, to make up carload lots.

YELLOW FEVER VACCINATIONS COMPLETED IN PUERTO RICO

Inoculation of the entire command at Puerto Rico against yellow fever has been completed without a single case of "unusual reaction," the War Department announced July 2. It was the largest mass yellow fever inoculation ever attempted in the history of the United States Army.

Decision to adopt this method of protecting troops in tropical regions against yellow fever authorized earlier this year grew out of the discovery by medical science of a jungle type of the fever that has its reservoir of virus in wild life as contrasted with malaria which is transmitted only between human beings. It cannot be stamped out and immunization was adopted in order to protect soldiers against possible infection in tropical regions.

All military personnel stationed at posts in the tropical regions of the Western Hemisphere are being inoculated against yellow fever. Troops ordered to duty in these regions will be vaccinated before leaving the states.

The yellow fever vaccine used by the Army consists of a special strain of yellow fever virus that has been attenuated through prolonged cultivation in tissue cultures by the Rockefeller Institute for Medical Research, New York. Extreme care is necessary in the packing and shipping of the vaccine, since it must be carried in containers in which the temperature does not exceed 39.2 F.

INDUSTRIAL HYGIENE IN DEFENSE INDUSTRIES

Approximately $250,000 is to be made available to the Division of Industrial Hygiene of the National Institute of Health for the 1942 fiscal year in order to continue the industrial hygiene activities in defense industries which were initiated on March 1 of this year. This amount, plus the regular appropriation of the division, will bring the entire budget for industrial hygiene in the Public Health Service close to $500,000. At present there are 106 persons engaged in this work, of whom 17 are physicians, 16 are engineers, 32 are chemists, toxicologists and physicists, and 41 are subprofessional and clerical workers. There are now 32 professional persons engaged directly in field activities in defense industries. This group consists of 12 physicians, 15 engineers, 4 chemists and 1 nurse. It is planned to increase this mobile field group in order to satisfy the demands for personnel in the states.

CANADIAN DOCTORS FOR BRITAIN

Arrangements have been completed between the British Red Cross and the Canadian Red Cross to send Canadian physicians to Britain. The project is comparable to the one which is sending American physicians to aid Britain. Fifty Canadian physicians are now ready to sail and more will follow, depending on transport facilities.

ORGANIZATION SECTION

OFFICIAL NOTES

ADDRESSES BY OFFICIAL STAFF

DR. W. W. BAUER:
August 20—National Medical Association, Chicago.

DR. MORRIS FISHBEIN:
August 1, 10:30 p. m. eastern daylight saving time—Listen America, Radio Station WEAF, Red Network of the National Broadcasting Company.
August 18—National Association Boards of Pharmacy and American Association Colleges of Pharmacy, Detroit.
August 25—Rotary Club, Toledo, Ohio.

DR. PAUL A. TESCHNER:
August 26—Teachers' Institute, Vincennes, Ind.
August 27—Teachers' Institute, Washington, Ind.

EXHIBITS FROM HEADQUARTERS

August 1-30.—County Fairs, California (in cooperation with California Medical Association):
The Respiratory System in Health and Disease.
Heart Disease.
Dangers of Self Diagnosis and Self Medication.
"Patent Medicine" Testimonials.

August 1-28.—County Fairs, Nebraska (in cooperation with Nebraska State Department of Health):
The Human Factory.
Prevention of Accidents.
Prevention of Burns.

August 1-30.—Toledo Museum of Science, Toledo, Ohio (in cooperation with Medical Society):
Medical Discoveries of a Century.

August 1-30.—Grand Rapids Public Museum, Grand Rapids, Mich. (in cooperation with Kent County Medical Society):
Heroes of Medicine.

August 1-30.—Cleveland Health Museum, Cleveland:
Your Bones and Muscles.

August 4-9.—Tri-State Fair, Burlington, Iowa (in cooperation with Des Moines County Health Unit):
The Public Health.

August 5-11.—Northern Wisconsin District Fair, Chippewa Falls, Wis. (in cooperation with Medical Society):
Periodic Health Examinations.

August 7-10.—Ramsey County Fair, White Bear, Minn. (in cooperation with Minnesota Society for the Prevention of Blindness):
Prevention of Eye Injuries.

August 9-17.—Illinois State Fair, Springfield, Ill. (in cooperation with Illinois State Department of Public Health):
Cancer.
Tularemia.

August 11-14.—Rock County 4-H Fair, Janesville, Wis. (in cooperation with Rock County Sanitary Unit):
The Human Factory.
Posture.

August 17-23.—American Pharmaceutical Association, Detroit (annual meeting):
Use and Abuse of Barbiturates.
Chemistry of the Sulfonamides.

August 18-23.—National Medical Association, Chicago (annual meeting):
Information About Syphilis.

August 24-30.—Minnesota State Fair, St. Paul (in cooperation with Minnesota Society for the Prevention of Blindness):
Prevention of Eye Injuries.

August 27-30.—National Woman's Christian Temperance Union, Grand Rapids, Mich. (annual meeting):
"Patent Medicines."

August 31-September 5.—Nebraska State Fair, Lincoln (in cooperation with Nebraska State Medical Society and Nebraska State Department of Health):
Information About Health.
The Human Factory.
Periodic Health Examinations.
Prevention of Accidents.
The Public Health.
Information About Syphilis.
Prevention of Burns.

August 30-September 5.—Indiana State Fair, Indianapolis (in cooperation with Indiana State Medical Society):
Cancer.
Tularemia.

MEDICAL LEGISLATION

MEDICAL BILLS IN CONGRESS

Changes in Status.—S. 415 has passed the Senate, providing pensions to members of the Regular Army, Navy, Marine Corps and Coast Guard who become disabled by reason of their service, equivalent to 90 per cent payable to war veterans for similar service connected disabilities. S. Res. 128 has been agreed to, directing the Railroad Retirement Board to make an immediate, thorough and complete investigation of the incidence of injuries and diseases incurred by employees through employment in the railroad industry and the social and economic consequences of such injuries and diseases, and to report to the Senate Committee on Interstate Commerce at the earliest practicable time the results of its investigation. H. R. 4476 has passed the House, providing for sundry matters affecting the Military Establishment. This bill includes an authorization under which the Secretary of War may provide for the "employment of interns who are graduates of or have successfully completed at least four years' professional training in reputable schools of medicine or osteopathy in the Medical Department, at not to exceed $720 per annum."

Bills Introduced.—S. 1711, introduced by Senator Reynolds, North Carolina, proposes to amend an act to provide for the promotion of vocational rehabilitation of persons disabled in industry or otherwise by making the act applicable "to any person inducted into the land or naval forces of the United States under the provisions of the Selective Training and Service Act of 1940 or ordered into the active military service of the United States under the provisions of the joint resolution of Aug. 27, 1940, as amended, who is disabled as the result of any injury sustained or disease contracted while in the military or naval forces of the United States." S. 1784, introduced by Senator Wheeler, Montana, proposes to enact a federal workmen's compensation act by providing compensation for disability or death resulting from injury to employees of contractors on public buildings and public works and to employees employed at places subject to the exclusive jurisdiction of the United States. The benefits of the Longshoremen's and Harbor Workers' Compensation Act would be made applicable, with certain limitations, to the employees covered by the pending bill. H. J. Res. 206, introduced by Representative Weiss, Pennsylvania, provides that the Civil Service Commission shall accept a report of the physical condition of any disabled war veteran made by any licensed physician on the basis of the examination given the disabled war veteran "within the last thirty days." H. R. 5414, introduced by Representative Jarman, Alabama, proposes an annual appropriation not to exceed $600,000,000 to provide for the health and welfare of children and to contribute to the future national defense by providing all children of the United States, of the age of 14 or less, with an adequate supply of wholesome milk.

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION AND PUBLIC HEALTH.)

ARIZONA

Navajo Clinical Conference.—The sixth Harlow Brooks Memorial Navajo Clinical Conference will be held at the Sage Memorial Hospital, Ganado, August 25-27. The following program has been announced:

Dr. Chevalier L. Jackson, Philadelphia, Cancer of the Esophagus.
Dr. James P. Rigg, Grand Junction, Colo., Upper Respiratory Foreign Bodies.
Dr. Clarence E. Rees, San Diego, Modified Technic for Amputation of the Lower Third of the Femur.
Dr. Louis F. Bishop Jr., New York, Harlow Brooks.
Dr. E. Payne Palmer, Phoenix, Recent Advances in Carcinoma.
Dr. Franklin I. Ball, Los Angeles, Mucous Membrane Lesions.
Dr. William W. Hutchinson, Los Angeles, Cyclopropane, a Safe and Effective Anesthetic.
Dr. Rupert B. Raney, Los Angeles, Technical Advances Having a Favorable Influence on Morbidity and Mortality from Intracranial Surgery.
Dr. Forrest N. Anderson, Los Angeles, Some Mental Viewpoints on Children's Problems.
Dr. Arthur S. Risser, Blackwell, Okla., The Finite in Medical Practice and Its Relation to the Infinite.
Dr. William H. Daniel, Los Angeles, Benign and Malignant Tumors of the Lower Colon.
Dr. Edwin Forrest Boyd, Los Angeles, Why Laugh?
Dr. Steele F. Stewart, Los Angeles, Tenosynovitis.
Dr. Newell Jones, Los Angeles, The Problem of Nutritional and Other Developments in Childhood.
Dr. Alfred E. Gallant, Los Angeles, Some Observations from Orthopedic Examinations of Men Under the Selective Service Act.
Dr. Tracy O. Powell, Los Angeles, Urological Problems in Children.
Dr. Buell H. Sprague, Los Angeles, Hypoglycemia.
Comdr. Joseph Madison Greer, Phoenix, medical corps, U. S. Naval Reserve, Surgery in the United States Navy.

CALIFORNIA

Lectures on Ophthalmology.—Dr. William Thornwall Davis, Washington, D. C., lectured before the Los Angeles Society of Ophthalmology and Otolaryngology, July 26, on "Differential Diagnosis of Squint and the Phorias with Particular Reference to the Value of Orthoptic Training"; "Treatment of Accommodative Squint," and "Disorders of Vertical Motility."

Postgraduate Courses.—Stanford University School of Medicine, San Francisco, announces a series of postgraduate medical courses for practicing physicians to be given in cooperation with the city department of health and the San Francisco Hospital, September 8-12. Each physician may take one morning and one afternoon course, or a full day course, and all physicians are urged to attend the evening general meetings. Morning courses will cover gynecology, medical diagnosis and treatment and diseases of the genitourinary tract, and afternoon courses the diagnosis and treatment of malignant tumors, cardiovascular diseases and surgical anatomy and operative technic. Full day courses will be on surgical emergencies, traumatic injuries and fractures, ophthalmology and anesthesiology. The evening meetings will be addressed by:

Dr. William H. Northway, Use of Physical Therapy in Modern Medicine.
Drs. Hale F. Shirley and Mary H. Layman, Behavior Problems in Pediatric Practice.
Drs. Rodney R. Beard, Nelson J. Howard and William P. Shepard, Everyday Problems in Industrial Medicine.

CONNECTICUT

Laboratory Technician Wanted.—An examination to fill the position of laboratory technician in a state laboratory will be held orally on August 25, and if a written examination is deemed necessary it will be held the same day. Applications must be filed by August 12. Candidates may offer not less than three years of experience in technical laboratory work or graduation from college with a course of study which included courses in chemistry and biology and not less than six months of experience in technical laboratory work; or graduation from high school and completion of an approved training course as a laboratory technician; or a satisfactory equivalent combination of experience and training. Candidates should have knowledge of standard laboratory practices, technics, equipment and terminology; ability to carry out detailed written and oral instructions; ability to keep standard laboratory records and to prepare reports of tests and examinations. The Connecticut residence is waived for this position, but candidates must be citizens of the United States. Entrance salary will be $1,440 annually less maintenance, with the maximum to be $1,860 annually less maintenance. Additional information may be obtained from the Personnel Department, State of Connecticut, State Capitol, Hartford.

ILLINOIS

New Medical Director of Industrial Commission.—Dr. Martin G. Luken, Chicago, has been appointed medical director for the Illinois Industrial Commission. Dr. Luken, a graduate of the University of Illinois College of Medicine, Chicago, was health officer of Chicago from 1906 to 1910. He is on the staff of St. Elizabeth's, Cook County and the Angel Guardian Orphanage hospitals. He succeeds Dr. Andrew Cosmas Garvy, Chicago.

Advisory Committee on Public Assistance.—At the request of the division of public assistance of the state department of public welfare the Illinois State Medical Society recently appointed a committee on medical care of public assistance recipients. Those appointed were Drs. Charles H. Phifer, Chicago, chairman; Harold M. Camp, Monmouth, secretary; Everett P. Coleman, Canton; Julius H. Hess, James H. Hutton and John R. Neal, Chicago. Dr. Edwin S. Hamilton, Kankakee, has been appointed to succeed Dr. Neal, who died July 1.

Poliomyelitis in Illinois.—Of the 56 cases of infantile paralysis reported in Illinois from the first of the year up to July 24, 26 occurred in Cook County and 30 downstate, the state department of health has announced. In a statement to the press, July 24, the department reported that only 3 new cases had been recorded during the first half of the week. In 1937, the peak epidemic year, it was exactly at this season that the number of cases mounted with totals of from 26 to 32 cases reported each week. A conference to outline a program of research work in infantile paralysis was held at the headquarters of the American Medical Association July 22, attended by local health authorities and representatives of the state department of health.

Chicago

Society News.—The Chicago Urological Society was addressed recently by Drs. Robert H. Herbst and James W. Merricks on "The Pathology of Vesical Neck Obstructions in the Female"; Leander W. Riba, "Chronic Urethritis in Women," and Russell D. Herrold, "Observations on the Use of Stilbestrol in Urology." Dr. Norris J. Heckel was chosen president of the society and Dr. Charles G. Weller, Aurora, vice president. Dr. Irving J. Shapiro is the secretary-treasurer.

Personal.—Dr. Anton J. Carlson, Frank P. Hixon distinguished service professor emeritus of physiology, University of Chicago, delivered the commencement address at the Massachusetts Institute of Technology on June 8.——Dr. James B. Herrick has been elected an honorary member of the Cardiac Society of Great Britain and Ireland.——Dr. Louis Leiter has resigned as associate professor of medicine at the University of Chicago, The School of Medicine, and has been appointed an attending physician at Michael Reese Hospital.

Postgraduate School of Tuberculosis.—Plans have been announced for the establishment of a Postgraduate School of Tuberculosis to offer two courses a year. It is expected that the school will be opened in October and may be held at any of the eight clinics of the Chicago Municipal Tuberculosis Sanitarium, a hospital or other location designated by the enrollees. Any branch or group supplying twenty or more enrolments can have the school established for their benefit at any suitable location and time convenient to the enrollees. The first twenty enrolments received will be given preference. The applications will be classified into "full registration groups," and the school will rotate from district to district following the sequence of the registrations. The plan to create the school was worked out by a committee of physicians and the board of directors of the Municipal Tuberculosis Sanitarium. Applications should be directed to the central office of the Sanitarium, 2049 Washington Boulevard.

INDIANA

Hospital News.—The new $1,000,000 "Silver Crest" Southern Indiana Tuberculosis Hospital was formally dedicated at New Albany recently. The hospital, which receives patients from forty southern Indiana counties, has a capacity of one hundred beds. Dr. Jerome V. Pace is superintendent.

Society News.—Dr. Paul B. Magnuson, Chicago, discussed "Treatment of Arthritis from the Standpoint of the General Practitioner" before the La Porte County Medical Society,

La Porte, June 19.——The Tippecanoe County Medical Society was addressed in Lafayette, June 10, by Dr. George B. Eusterman, Rochester, Minn., on diagnosis and treatment of chronic peptic ulcer and gastritis.——Dr. Carl P. Huber, Indianapolis, addressed the Pike County Medical Society, Petersburg, July 10, on obstetrics.——William Crawford, D.D.S., Indianapolis, addressed the Dearborn-Ohio County Medical Society, Dillsboro, June 5, on "Medicine and Dentistry."

KENTUCKY

Society News.—The Four County Medical Society, composed of Caldwell, Lyon, Trigg and Crittenden counties, held a pediatric conference at its quarterly meeting in Princeton, July 8, with Drs. Philip F. Barbour and Lee Palmer, Louisville, as guest speakers. Dr. Barbour discussed "Abdominal Pain in Children" and Dr. Palmer "Diarrhea and Dysentery."

Epidemic of Shiga Dysentery.—The American Red Cross has established a twenty-five bed emergency hospital in a school building at Columbia, Adair County, to serve as an isolation unit in an epidemic of Shiga dysentery. About 100 cases have been reported with twelve deaths. Children are the chief victims, it was said. The emergency hospital was set up on the recommendation of Dr. Lydia B. Edwards, Baltimore, who made a survey for the Red Cross and state health authorities. Houses will also be quarantined in an effort to curb the disease. A staff of seven Red Cross nurses and one Red Cross doctor and five additional nurses is being recruited in the vicinity, according to an announcement from the Red Cross.

LOUISIANA

Personal.—Drs. Joseph O. Weilbaecher Jr. and Charles B. Odom, who until recently had been acting director and assistant director, respectively, of Charity Hospital, New Orleans, were guests at a banquet in their honor given by members of the staff. They resigned to enter private practice.

Graduate Course in Tropical Medicine.—The department of graduate medicine, Tulane University of Louisiana School of Medicine, New Orleans, announces a graduate course in tropical medicine and medical parasitology. The course will consist of lectures, conferences, quizzes, practical laboratory work and demonstrations, together with the study of clinical cases, with about half of the time devoted to diagnosis and management of disease. The course will begin each fall in September with the opening of the medical school and extend through the first half of the school year. It is open to properly qualified American physicians and adequately trained physicians in Latin America and other parts of the world. Additional information may be obtained from the Director, Department of Graduate Medicine, Tulane University of Louisiana School of Medicine, 1430 Tulane Avenue, New Orleans.

MAINE

State Medical Election.—Dr. Carl H. Stevens, Belfast, was chosen president-elect of the Maine Medical Association at its annual meeting in York Harbor, June 22-24, and Dr. Penry L. B. Ebbett, Houlton, was inducted into the presidency. Dr. Frederick R. Carter, Augusta, is the secretary-treasurer and business manager.

State Board Changes.—Dr. Franklin A. Ferguson, Portland, was elected chairman of the state board of registration in medicine at the July meeting and Dr. Adam P. Leighton, Portland, secretary. Dr. Magnus F. Ridlon, Bangor, was recently appointed a member to succeed Dr. George R. Hagerthy, Bar Harbor, whose term expired.

Society News.—Dr. Siegfried J. Thannhauser, Boston, addressed the Aroostook County Medical Society in Houlton, June 10, on "X-Ray Therapy in Treating Skin Diseases and Super Growths."——Dr. Gilbert E. Haggart, Boston, addressed the Piscataquis County Medical Society, Greenville, July 24, on "Low Back Pain with Special Reference to Herniation of Intervertebral Disks."

MICHIGAN

Honorary Memberships Conferred.—The Wayne County Medical Society recently conferred honorary membership on three persons for outstanding service in their respective fields: Roy C. Gamble, portrait artist; Gustavus D. Pope, for twenty-one years a member of the board of commissioners of the Detroit Department of Health and since 1917 chairman of the Detroit chapter of the American Red Cross, and Milton S. Van Geison, president of the Michigan Association of Superintendents of the Poor and director of the Flint Public Welfare Board.

New Center for Study of Brain.—Announcement has been made of the establishment of the Wayne University Brain Disease Registry at Wayne University College of Medicine, Detroit, in cooperation with seventeen hospitals in the Detroit metropolitan area. The center will be under the auspices of the department of pathology at the university and under the direction of Dr. Gabriel Steiner, professor of neurology and neuropathology. It is planned to make the facilities of the brain registry available to physicians and other scientists without charge. According to newspaper reports, its services will include storage of tissue specimens for examination and study, reports and statistics on the examined cases, teaching and demonstration facilities, publications and neuropathologic research.

Changes in Health Officers.—Dr. Alexander Witkow, Brooklyn, has been placed in charge of the health unit in Dickinson County, succeeding Dr. Edwin H. Place, now of Montgomery, Ala., resigned.——Dr. Vida H. Gordon, Lansing, has been appointed director of the Sanilac County health department, effective June 2.——Dr. Clifford C. Corkill, Menominee, was to resume his position as director of the Menominee County health department June 1 after a nine months leave of absence to study for a master's degree in public health at Johns Hopkins University School of Hygiene and Public Health. Dr. Buell H. Van Leuven, Petoskey, who was acting director during Dr. Corkill's absence, is now director of the Grand Traverse County health department. ——Dr. August C. Orr, Newberry, resigned as director of the Luce-Mackinac district health department recently to enter private practice in Bismarck, N. D.——Dr. Nelson J. Robbins, Negaunee, has been named city health officer. During the thirty-seven years Dr. Robbins has practiced in Negaunee, he has served four terms as mayor and several years as health officer.

MINNESOTA

Hospital News.—An expansion program, to be worked out over several years at an ultimate cost of between $150,000 and $200,000, is under way at St. Joseph's Hospital, St. Paul. Recently the hospital acquired the Capitol Annex, an apartment building at St. Peter and Exchange streets, and a tract with an 80 foot frontage on Tenth Street adjoining the hospital properties on that street.

Society News.—Dr. Gustaf T. Nordin, Minneapolis, was elected president of the Minnesota Radiological Society at its annual meeting in St. Paul, May 27; Dr. Arthur U. Desjardins, Rochester, vice president, and Dr. John P. Medelman, St. Paul, was reelected secretary.——Major Harry G. Armstrong, flight surgeon, medical corps, U. S. Army, gave a Mayo Foundation lecture in Rochester, July 1, on "Recent Advances in Aviation Medicine."

MISSOURI

Personal.—Dr. Ernest Sachs, professor of clinical neurologic surgery, Washington University School of Medicine, St. Louis, has been elected an honorary member of the Royal Society of Medicine of England.——Dr. James W. Chapman, Jefferson City, was recently chosen president of the Missouri Public Health Association.

New Health Officers.—Dr. Edward G. McGavran, health officer of Monongalia County, W. Va., and director of a health training center of the state health department at Morgantown, has been appointed health officer of St. Louis County. He succeeds Dr. Theodore R. Meyer, who had served since 1936. Dr. Meyer has entered active service in the U. S. Navy at Corpus Christi, Texas.

NEW JERSEY

Personal.—Dr. George O'Hanlon, medical director of the Jersey City Medical Center, has been appointed medical director of all public hospitals in Hudson County, by action of the Hudson County freeholders and the city commission of Jersey City. The action was taken under the authority of a new state law allowing first class cities and first class counties to appoint a medical director for all city and county hospitals. Dr. Samuel A. Cosgrove, director of Margaret Hague Maternity Hospital, Jersey City, has been appointed deputy director. ——Dr. Clarence W. Way, Sea Isle City, secretary of the Cape May County Medical Society, a major in the U. S. Army Medical Reserve Corps, has been ordered to active duty at Fort Dix.

NEW MEXICO

State Medical Election.—Dr. Wallace P. Martin, Clovis, was chosen president-elect of the New Mexico Medical Society at the annual meeting in Raton, May 26, and Dr. Carl Mulky, Albuquerque, was installed as president. Dr. Joseph E. J. Harris, Albuquerque, was elected vice president. The next annual session will be held in Santa Fe.

NEW YORK

Society News.—Dr. James H. Flynn, Troy, was elected president of the New York State Health Officers Association at the annual meeting, June 25; Drs. Myron M. Metz, Williamsville, William H. Runcie, Freeport, and John R. MacElroy, Jonesville, were elected vice presidents, and Dr. Russell H. Wilcox, Tonawanda, was made secretary.——Dr. Robert R. Linton, Boston, addressed the Saratoga County Medical Society at the Saratoga Spa, June 19, on "Diagnosis and Treatment of Peripheral Vascular Disease."——Drs. Lucius A. Wing and Joseph E. J. King, New York, addressed the Chemung County Medical Society, Elmira, in June on "Recent Advances in Obstetrics" and "Head Injuries" respectively.

Bacillary Dysentery in a Nursing Home.—An outbreak of bacillary dysentery (Sonné) in a nursing home in Suffolk County is reported in *Health News*, July 21. The outbreak began in the babies' ward and then spread to other parts of the institution, affecting 60 of the 275 inmates. Six of the 38 babies died. A diet kitchen in the babies' ward prepared formulas for the children. It was believed that opportunity for contamination existed in the handling of this formula. Another clue was found in the mother of one of the children who visited the home the day before the outbreak. The woman lived in a home near the institution for a two day visit and during that time complained of diarrhea, it was said. Epidemiologic investigation was still in progress.

New York City

Presbyterian Hospital to Benefit from Bequest.—The late Arthur Curtiss James, railroad industrialist, bequeathed nearly eight tenths of his extensive estate for the establishment of the James Foundation, the income of which is to be used to aid various charitable, educational and religious institutions. The fund is to be divided into twenty-eight shares, of which Presbyterian Hospital is to receive two shares, according to the will.

Honorary Degrees.—Dr. John J. Moorhead, consulting surgeon, New York Post-Graduate Hospital, and Dr. George B. Wallace, professor of pharmacology and director of the pharmacologic laboratories, New York University College of Medicine, received the honorary degree of doctor of science at the commencement of New York University. The doctorate of public health was conferred on Drs. William E. Caldwell, professor of clinical obstetrics and gynecology at Columbia University College of Physicians and Surgeons, and Frederick C. Holden, professor emeritus of obstetrics and gynecology, New York University College of Medicine.

Faculty Changes at New York Medical College.—Dr. Loudon Corsan Reid, assistant professor of pathology at New York Medical College, has been transferred to the department of physiology. Dr. Stephen P. Jewett, professor of psychiatry, has been made head of a new department of psychiatry and Dr. Thomas I. Hoen, professor of neurosurgery, head of the department of neurology and neurosurgery. Dr. Joseph H. Fobes, professor of surgery, has been placed in charge of graduate surgery and Dr. Louis Rene Kaufman, professor of surgery, in charge of undergraduate surgery. Dr. Reuel A. Benson, professor of pediatrics, now has the title of Helen S. Case professor of pediatrics.

NORTH CAROLINA

Personal.—Dr. William Redin Kirk, Hendersonville, celebrated his fiftieth anniversary in the practice of medicine, June 19.——Dr. William G. Cheves, Bunn, has been appointed medical director of the state prison system and Dr. Benjamin J. Lawrence, Raleigh, prison surgeon.——Dr. Len D. Hagaman, Boone, has been appointed health officer of Caldwell and Burke counties to succeed Dr. Wesley G. Byerly, Lenoir, who resigned because of ill health.

District Meeting.—The Tenth District Medical Society held its spring meeting in Waynesville recently with Dr. Oscar L. Miller, Charlotte, as the guest speaker on "Elbow Injuries, Particularly Those Fractures in Which Immediate Open Operation Is Indicated." Other speakers included Drs. Daniel L. Smith Jr., Spartanburg, S. C., on "Recent Advances in Infant Nutrition"; Samuel L. Crow, Asheville, "Diagnosis and Treatment of Cardiac Emergencies," and James S. Brown, Hendersonville, "Patience in Obstetrics."

NORTH DAKOTA

State Medical Election.—Dr. Alfred R. Sorenson, Minot, was named president-elect of the North Dakota State Medical Association at the annual meeting in May at Grand Forks and Dr. Frederick W. Fergusson, Kulm, became president. Drs. Frank I. Darrow, Fargo, and Arne O. Arneson, McVille, were elected vice presidents and Dr. Leonard W. Larson, Bismarck, was reelected secretary. The 1942 meeting will be in Jamestown.

OHIO

Personal.—Dr. Carl J. Wiggers, Cleveland, received the honorary degree of doctor of science from the University of Michigan at the commencement, June 21.——Dr. James W. Rowe, at various times demonstrator in histology, instructor and adjunct professor of obstetrics and assistant professor of gynecology at the University of Cincinnati College of Medicine, was honored at a testimonial dinner recently. About one hundred and fifty physicians attended and presented Dr. Rowe with a painting.

Society News.—Officers of the Alumni Association of the University of Cincinnati College of Medicine elected at the recent annual meeting are Drs. Daniel C. Rivers, president; Irving H. Schroth, vice president, and Helena T. Ratterman, secretary.——Drs. Arthur A. Brindley and Frederick W. Clement, Toledo, addressed the Auglaize County Medical Society, June 26, on "Spinal Anesthesia, Surgical and Therapeutic" and "Oxygen Therapy" respectively.——Dr. Donald M. Glover, Cleveland, discussed "Modern Treatment of Burns" before a joint meeting of the Miami and Shelby county medical societies in Sidney, June 12.——Dr. Henry Kennon Dunham, Cincinnati, was elected president of the Ohio Public Health Association at the annual meeting in Columbus, May 22.

SOUTH CAROLINA

Public Health Meeting.—Dr. Joseph E. Brodie, Greenwood, was named president-elect of the South Carolina Public Health Association at the recent annual meeting at Myrtle Beach, and Dr. Alva W. Humphries, Camden, became president. Speakers included Dr. Halbert L. Dunn, Washington, D. C., chief statistician for vital statistics, U. S. Bureau of the Census, on birth and death registration; Col. William H. Moncrief, medical superintendent of the South Carolina Sanatorium, State Park, on hospital facilities and treatment of tuberculosis in the state, and Dr. James G. Townsend, chief of the division of industrial hygiene, National Institute of Health, Bethesda, Md., on safeguarding the health of workers.

TENNESSEE

Poliomyelitis Increases.—Thirty cases of poliomyelitis have been reported in an outbreak in Franklin and Coffee counties, according to a newspaper dispatch, July 27. A report on July 24 said that three deaths had occurred. The U. S. Public Health Service has assigned a member of its staff to assist state health officers to curb the spread of the disease, it is reported.

WASHINGTON

State Medical Meeting in Seattle.—The fifty-second annual meeting of the Washington State Medical Association will be held in Seattle at the Olympic Hotel, August 24-27, under the presidency of Dr. Homer D. Dudley, Seattle. Washington physicians will present the scientific program on Tuesday and Wednesday. Morning sessions will be devoted to presentation of papers on general subjects; in the afternoons there will be panel discussions and symposiums. Among the papers will be:

Dr. Leo J. Rosellini, Seattle, Practical Applications of Use of Large Quantities of Blood Plasma.
Dr. Edwin G. Bannick, Seattle, General Status of Chemotherapy.
Drs. Lester J. Palmer and George D. Capaccio, Seattle, Present Day Insulins.
Dr. Roy J. Popkin, McChord Field, Diagnosis and Treatment of Peripheral Vascular Diseases.
Dr. John C. Lyman, Walla Walla, Treatment of Chronic Massive Ulcerative Infections of the Skin with Zinc Peroxide.
Dr. James M. Nelson, Spokane, Some Physiologic Aspects Relating to Surgery.

Subjects and chairmen of the symposiums will be: traumatic conditions, Dr. Raymond L. Zech, Seattle, Tuesday, and Dr. Robert D. Forbes, Seattle, Wednesday; heart conditions, Dr. Charles E. Watts, Seattle; infantile paralysis, Dr. Burton A. Brown, Tacoma, and diseases of ear, nose and throat, Dr. Har-

old LeRoy Goss, Seattle. Panel discussions will be held on head injuries, with Dr. Hale A. Haven, Seattle, as chairman; obstetrics, Dr. Richard D. Reekie, Spokane, chairman, and pediatrics, Dr. Jay I. Durand, Seattle, chairman.

WISCONSIN

State Board Election.—Dr. Donald R. Searle, Superior, was elected president of the Wisconsin State Board of Medical Examiners at a meeting in Milwaukee, June 26, and Dr. Robert E. Flynn, La Crosse, secretary.

Fifth District Meeting.—At a meeting of the Fifth Councilor District of the State Medical Society of Wisconsin in Sheboygan, June 12, the speakers included Drs. John M. Waugh, Rochester, Minn., on "Vaginal Hysterectomy"; Walter C. Alvarez, Rochester, "Puzzling Types of Abdominal Pain," and William S. Middleton, Madison, "Use and Abuse of Some Common Drugs." A symposium on cardiovascular disease was presented by Drs. Sherburne F. Morgan, Maurice A. F. Hardgrove, Howard L. Correll and Robert H. Feldt, all of Milwaukee. Following a banquet Dr. Ralph P. Sproule, Milwaukee, president of the state society, made an address and Dr. Alvarez spoke on "Why Women Get Nervous."

PUERTO RICO

Dr. Wegman Named Assistant Professor at School of Tropical Medicine.—Dr. Myron E. Wegman, since 1936 pediatric consultant of the bureau of child hygiene of the Maryland State Department of Health, Baltimore, is serving as assistant professor of child hygiene in the School of Tropical Medicine of the University of Puerto Rico, under the auspices of Columbia University, New York, and as assistant director of the division of education and research in charge of child hygiene in the insular department of health. Dr. Wegman's new work will include instruction in child hygiene for health officers and public health nurses and postgraduate instruction in pediatrics for physicians. He will also serve as consultant in pediatrics to the University Hospital and the Bayamon District Hospital and will carry on research in child hygiene and pediatrics. His headquarters will be at San Juan. Dr. Wegman served on the pediatric staff of the New Haven Hospital and Yale medical school from 1932 to 1936 and attended part time sessions at Johns Hopkins University School of Hygiene and Public Health, Baltimore, from 1936 to 1938, when he received the degree of master of public health. He has been lecturer in public health administration at the school of hygiene and public health, in charge of the course in child hygiene, during the last two years.

GENERAL

Ask Health Funds for Defense Areas.—Dr. Thomas Parran, surgeon general, U. S. Public Health Service, Washington, D. C., told a committee of the House of Representatives studying defense migration, July 18, that an additional $1,885,137,891 would be needed to provide health facilities in "preparedness areas," according to an Associated Press dispatch. Of this amount Dr. Parran said $1,524,436,000 should be spent for housing and $170,874,294 would be needed for medical care.

Appendicitis Death Rate Reduced.—The death rate from appendicitis among industrial policyholders of the Metropolitan Life Insurance Company has dropped from 14.4 per hundred thousand in 1929 to 8.9 in 1940, the company recently announced. The decrease was attributed to improved diagnostic and surgical technic, and to educational campaigns that have taught the public to seek prompt medical attention. It is believed that the greatest achievement of the campaigns has been in spreading the warning against taking laxatives in the presence of abdominal pain.

National Bowling Medical Group Formed.—Officers were elected for the American Medical Bowling Association during its first representative group meeting in Cleveland in June. Dr. Lewis Wine Bremerman, Los Angeles, was chosen president; Dr. James Robert Smith, Erie, Pa., vice president, and Dr. Maurice L. Allen, Cleveland, Ohio, secretary-treasurer. First place in this year's competition, the first, went to a team from Erie composed of Drs. Archie J. DeSantis, James R. Smith, Frank L. Mozdy, Donald D. Williams and Frank J. Theuerkauf. This team also placed first in the doubles, singles and the all events. The winning trophy was donated by Mead Johnson & Company, Evansville, Ind.

Report on President's Birthday Celebration for Poliomyelitis Campaign.—The National Committee for the Celebration of the President's Birthday made its report on the 1941 celebration to the President on July 23. The observances of last January 30 raised a net total of $2,104,460.53, which exceeded the amount raised in 1940 by $697,214.79. The net proceeds will be divided between state and local chapters for direct relief to victims and the central organization for use in combating epidemics, in promoting research and in aiding hospitals and institutions in the study and treatment of the disease. State and local chapters will receive $1,096,865.84 and the foundation $1,007,594.69. Expenses of the campaign were reported as $136,996.61, or 6.1 per cent of the total.

Special Society Elections.—Dr. Hunter H. McGuire, Winchester, Va., was named president-elect of the American Ophthalmological Society at the annual meeting, May 28, in Hot Springs, Va., and Dr. Allen Greenwood, Boston, was installed as president. The next annual session will also be at Hot Springs.——Dr. Thomas E. Carmody, Denver, was elected president of the American Laryngological Association at the annual meeting in Atlantic City, May 28, and Dr. Charles J. Imperatori, New York, was reelected secretary.——At the first annual meeting of the American Diabetes Association in Cleveland in June Dr. Herman O. Mosenthal, New York, was elected president; Drs. Joseph T. Beardwood, Jr., Philadelphia, and Joseph H. Barach, Pittsburgh, were elected vice presidents and Dr. Cecil Striker, Cincinnati, was made secretary.— Dr. Edwin T. Wyman, Boston, was elected president of the American Association of Medical Milk Commissions at the annual meeting in Cleveland, June 3, and Dr. Paul B. Cassidy, Philadelphia, was reelected secretary.——Miss Mary Louise Marshall, librarian of the Tulane University of Louisiana School of Medicine, New Orleans, was elected president of the Medical Library Association at the annual meeting recently in Ann Arbor, Mich. Dr. John F. Fulton, New Haven, Conn., was elected vice president and Miss Anna C. Holt, librarian of Harvard Medical School and the Harvard School of Public Health, Boston, was reelected secretary. The 1942 convention will be held in New Orleans.

LATIN AMERICA

Medicodental Meeting in Mexico.—The third medicodental convention organized by the Mexican Association of Orthodontia was held in Mexico City, June 23-28. Among physicians on the program were:

Dr. Henry Borsook, Pasadena, Calif., Vitamins in Prophylaxis and Therapy.
Dr. Ignacio Chavez, Mexico City, Dental Focal Infection and Cardiac Rheumatism.
Dr. Eberle Kost Shelton, Los Angeles, Endocrine Factors in Growth and Development.
Dr. Francisco de P. Miranda, Mexico City, Importance of Mineral Salts in the Diet of the Child and the Adolescent.
Dr. Antonio Torres Estrada, Mexico City, Improved Technic for Dacryocystorhinostomy.
Dr. Hermann Becks, San Francisco, Endocrinology.
Dr. Miguel E. Bustamante, Mexico City, General Hygiene and Dental Hygiene.

CANADA

Tuberculosis Meeting.—Dr. Duncan A. Carmichael, Ottawa, was named president-elect of the Canadian Tuberculosis Association at the annual meeting in Toronto in June and Dr. John H. Holbrook, Hamilton, became president. Dr. William Boyd, Toronto, was the guest speaker at a joint session with the Ontario Laënnec Society on "Bronchogenic Carcinoma."

FOREIGN

Prize for Research in Encephalitis.—The foundation at the University of Berne, Switzerland, to promote research on encephalitis lethargica announces its annual prizes. Those who wish to compete should send their applications to the Dean of the Medical Faculty, which allocates the prizes at the end of each year.

Hospital in Scotland Named for Paderewski.—Funds are being collected for a Polish hospital in Edinburgh, Scotland, to be established in connection with a medical school as part of the Polish Medical Center in Edinburgh, which has been in operation since March, the New York *Times* recently reported. The hospital will be named the Paderewski Hospital in Edinburgh, in honor of the late Ignace Jan Paderewski, pianist and Polish premier. The hospital will be open to Polish civilian refugees, to Polish soldiers in Scotland and, in an emergency, to the British public. A total of $20,000 has been sent from New York by the Paderewski Testimonial Fund for the hospital. A building has been made available by the city of Edinburgh, and the present drive is for a fund of $50,000 to buy equipment. The fund in honor of Paderewski was started before his death as a tribute on the occasion of the fiftieth anniversary of his American début.

Foreign Letters

LONDON

(From Our Regular Correspondent)

June 11, 1941.

The Destruction at the Royal College of Surgeons

The destruction by a German bomb of the larger part of the greatest medical museum in the world has been described in a previous letter. Further particulars can now be given. The lecture room, where discourses have been heard for a century from almost all the leaders of British surgery, is now a charred ruin. The main block of the library remains with books still on the shelves, but its state is too dangerous for use. Nearly ninety thousand volumes had been sent to various parts of the country after damage by blast from a previous raid. The transfer was aided by a grant from the Rockefeller Foundation. The principal treasures of the library had been sent away before the outbreak of the war. But the museum, which possessed treasures such as no other country could boast, has suffered irreparable loss, although many of the most valuable specimens were saved by having been placed in a tunnel below the basement surrounded with sand. The basement covers a wide area, and most of it escaped the fire that occurred. Many rooms, cellars and tunnels connected with it, which had been reinforced, were used for other specimens, which were not damaged. The important college records and historical documents had been sent away. But the working records dealing with the specimens, saved in the basement and subbasement, and the records of pathologic specimens presented to the college during the last few years were destroyed. The preservation of copies of the museum catalogues had been carefully considered and insured. All were saved except the catalogue of part of the pathologic section and that of the curio room, but nearly all the specimens in this room are described in the "Guide to Surgical Instruments and Objects in the Historical Series." Of the famous Hunterian collection, which forms the basis of the museum, no fewer than 3,750 specimens have been saved. Of the collections illustrating human anatomy only 20 specimens have been saved. Of 5,200 mammalian specimens illustrating comparative osteology only 20 or 30 remain and hardly any of 3,000 avian specimens or of the large amount of amphibian, reptile and aquatic material. The two rooms built in the middle of the last century, containing physiologic and comparative anatomy specimens, have been blasted away. Much anthropologic material has been destroyed, including the fine collection of primitive Tasmanian and Australian skulls. The greater part of the large collection of instruments is safe and can be restored. Among these are the instruments of Lister and of Moynihan, the Chinese and Japanese collections and the series illustrating the evolution of anesthetic apparatus. Though irreparable loss has been suffered, the destruction is not so great as was at first feared. Enough has been saved for the basis of a new museum, which will continue the Hunter tradition, which has always been fundamental in the college.

Changes Due to the War in National Health Insurance

The government has announced changes in the national health insurance scheme necessary to meet wartime conditions. It proposes to increase the sickness benefit for a man from $4 to $4.50 a week and his disablement benefit from $2 to $2.50. For single women and widows the new rates will be $4 and $2 respectively. These changes will be made possible by state grants on the usual basis and by increasing by 4 cents the weekly contributions payable in respect of each person, of which 2 cents will be borne by the employer and 2 cents by the insured person. The government also proposes to raise the income limit above which insurance is not compulsory for nonmanual workers from $1,250 to $2,120 per annum. This will increase by nearly half a millon the number of persons who will be entitled to medical treatment under the national health insurance scheme. The new position which will arise is being discussed with the medical profession. Power will be sought in the bill to be introduced to deal with the position in insurance of civilian prisoners of war and other similar war problems. The position of British prisoners who were rescued from the German raider *Altmark* is an example. Similar problems are arising in the course of the war.

Delay in Appointing to Hospitals Friendly Alien Physicians

There are in this country about fourteen hundred friendly alien physicians—seven hundred and fifty Germans, Austrians and Italians, two hundred and fifty Czechs and three hundred and fifty Poles. In January a statutory order was passed enabling alien physicians to be placed on a temporary register of the General Medical Council so as to be eligible for positions in hospitals and institutions. It was intended that the names submitted by various organizations should first be put before the Home Office for general permission to undertake such work. If approved, the names would be sent to the Aliens War Service Department and candidates for appointments would be finally selected by the Central Medical War Committee. The names would then be placed on the temporary register.

The machinery has worked very slowly. Since January only two hundred and fifty physicians have been approved by the Home Office and fewer than two hundred have been placed on the temporary register, and those who have obtained appointments have done so by answering advertisements in the press. But the government now seems alive to the importance of accelerating the process. These aliens are all refugees from totalitarian tyranny and anxious to help the Allied cause. Their dossiers have been for months with the Home Office, which has now agreed to deal with one hundred cases or more a week, but apparently the Aliens War Service Department can handle only fifty. If so, delay is inevitable. The slowness appears to be a question of adequacy of staff. The demand for physicians is so urgent that all obstacles should be removed. The great trouble is the possibility that among these aliens there may be enemy agents masquerading as friendly.

At a meeting of the Socialist Medical Association in London a resolution was passed protesting against "the government's failure to make use of the medical services of the anti-Fascist refugee doctors in this country." An immediate speed-up was urged. Mr. Somerville Hastings (laryngologist), president of the association, said that the state of affairs was more the fault of the government than of the British Medical Association. He had seen the Home Secretary twice on the matter and it had now been agreed to deal with one hundred applications weekly, but this rate was far too slow.

A New Use for Wounded Soldiers

In the House of Commons Mr. Churchill gave the reason why the mistake had been made of reporting officially that German parachutists landed in Crete in the uniforms of the New Zealand soldiers. After landing at one point the Germans drove a number of New Zealand walking wounded before them and along with them. It is well known that in the last war the Germans used civilians in this way in Belgium as a screen to protect their troops, but the use of wounded soldiers for this purpose appears to be quite new as a method of warfare.

Physician Bombed Out Four Times But Still Carries on

The London Insurance Committee commended a panel (national health insurance) physician who was bombed four times but was still carrying on. He had an office which was totally demolished last winter. He secured accommodations in the same street and six weeks later these premises were damaged beyond repair. Again he opened another office in the same

street, but this was demolished in the spring with all his furniture, drugs, bottles and medical records. His house in the neighborhood also was destroyed. He secured a fourth office in the same street and intimated to the committee his determination not to give up while a place remains. The committee sent him a message of sympathy and admiration.

BUENOS AIRES

(From Our Regular Correspondent)

May 22, 1941.

Congress for Care of Cardiac Patients

The Congress of the Asistencia Social del Cardiaco met in April in Buenos Aires to discuss problems relating to heart disease. Prof. Rafael A. Bullrich, professor of internal medicine in Buenos Aires, the chairman, pointed out that the stress of modern civilization greatly increases the morbidity and mortality from circulatory diseases. In the scientific sessions Bullrich discussed fundamental legislative points of view affecting social welfare of persons suffering from heart disease. He favored laws of social prophylaxis and welfare, recognition of cardiology as a medical and university specialty, a national ministry of public health and health records for school children from the time they entered school, and he stressed the need of old age pensions. He recommended the creation of dispensaries throughout Argentina for persons with heart disease and hospital colonies for the care and vocational training of children with heart disease. Dr. Luis González Sabathié of the University of Rosario said that the old methods of purely medical treatment in cardiac disorders were insufficient and that the quest for new therapies was stimulated by the recognition of the social significance of heart disease. Drs. José Maria Macera and Alberto Ruchelli, both pediatricians, recommended the establishment of special institutes for heart disease, such as are organized in the United States and Great Britain. They found that 8,500 school children (2.4 per cent) in Buenos Aires had heart disease. Of these, only 9.36 per cent received medical and hospital treatment. They also found that 0.64 per cent of all children in Argentina had asymptomatic primary heart disease, unrecognized at the time but disclosing itself subsequently. Dr. I. Urbandt discussed cardiovascular disturbances among industrial workers and recommended studies to determine activities suitable for affected industrial workers and regular periodic examinations. A considerable number of these workers could not be readjusted to other work. According to statistics, 70 per cent of revolutionists were persons who through physical disabilities, inclusive of heart disease, had become failures in life.

Resolutions were adopted requesting the government to legislate on social prophylaxis and welfare of patients with heart disease and to organize the medical care of such persons. Early diagnosis and long continuing treatments constitute the basis of social welfare for these patients. To this point of view the public needs to be educated.

Rheumatism in Children

In 1931 a cardiologic and rheumatologic division was organized in the Institute for Pediatrics in Buenos Aires to meet the increased incidence of rheumatic diseases in children and to enable their steady supervision. Its present director, Dr. A. Puglisi, recently reported on the activities of this institute. During the first ten months of 1940, 225 children, of whom only 6 were foreign children, were examined (122 boys and 103 girls). Fifty-two (23 per cent) of these children were hospitalized. The age level of these children varied between 3 and 16 years and was distributed as follows: 3 years 1, 4 years 7, 5 years 14, 6 years 18, 7 years 16, 8 years 29, 9 years 31, 10 years 24, 11 years 11, 12 years 18, 13 years 23, 14 years 20, 15 years 4, 16 years 1. The search for familial involvements showed that the father had heart disease in 9 cases and the mother in 16, a total of 11 per cent; likewise the father was rheumatic in 15 cases and the mother in 32, representing a total of 20 per cent. One hundred and fifty-one of the 225 children presented articular rheumatism with cardiac complications. Of these, 76 (33 per cent) could be cured of the complications but remained rheumatic. Eight of the 76 were affected with chorea. Of the 151 with endocarditis, 27 (19 per cent) recovered; in 117 (77 per cent) the condition remained unmodified. Seven (4 per cent) died, 3 of septic endocarditis and 4 of asystoly. Fifty-eight (76 per cent of the 76 rheumatic children without complications) regained health; no improvement was effected in 18 (24 per cent).

Marriages

CHARLES WHITE MILLENDER, Asheville, N. C., to Miss Dorothy Thompson Aiken of Columbia, Tenn., in Atlanta, Ga., June 7.

JOHN HAROLD SHANKLIN, Parkersburg, W. Va., to Miss Nancy Elizabeth Bedford of Osawatomie, Kan., July 5.

MARVIN DEAVER MCCULLOUGH, Knoxville, Tenn., to Miss Margaret Elizabeth Lane of Lone Mountain, May 1.

JAMES HARVEY JOHNSTON JR., Spartanburg, S. C., to Miss Jane Chauvin Hochendel in Houma, June 14.

ALBERT EARL BARNHARDT, Kannapolis, N. C., to Miss Margaret Helen Martin of Shelby in May.

HENRY BOONE GRANT, Garysburg, N. C., to Miss Elizabeth Cheatham Applewhite of Halifax, May 20.

SETH A. BRUMM, Philadelphia, to Mrs. Carver Havemeyer of New York in Middleburg, Va., in May.

MARLER SLATE TUTTLE, Kannapolis, N. C., to Miss Miriam Thompson Goodrum of Davidson, June 18.

EUGENE FREDERICK KOSTER, East Orange, N. J., to Miss Mary Jane Miller of Cape May, May 31.

FRANKLIN JOHNSON MOORE, Chicago, to Miss Helen Curry Stevenson of Columbia, S. C., in June.

CHARLIE LOUIS SYKES, Pilot Mountain, N. C., to Miss Dorabelle Graves at Mount Airy in May.

CHARLES WATERS THOMPSON, Washington, D. C., to Miss Betty Nichols in New York in June.

JEROME BOSTICK HAMER, Charlotte, N. C., to Miss Frances Rice Hill of Roanoke, Va., May 17.

VERNON BOULTER BEAM, Baton Rouge, La., to Miss Malcolm Thompson at New Orleans, May 11.

EDWARD LOWRY SHULER, State Park, S. C., to Miss Alberta Stoll Prince of Charleston in May.

EDWARD R. ANNIS, Tallahassee, Fla., to Miss Betty Starck of Miami in Jacksonville, June 16.

JOSEPH SAMUEL HOLBROOK, Statesville, N. C., to Miss Nancy Wheeler Cox of Raleigh in May.

WILLIAM LAMAR BRYAN JR. to DR. MARGARET E. STRANGE, both of New Orleans, May 30.

ERNEST HENRY ASCHENBACH to Miss Clara Chacouas, both of Washington, D. C., June 19.

JAMES GORDON MCMILLAN to Miss Willie Logan, both of Chattanooga, Tenn., in May.

CHARLES S. HERTZ, Allentown, Pa., to Miss Lillian Stokes at Charlotte, N. C., May 1.

GUSTAVE A. HAGGSTROM to Miss Margaret Fulton Cameron, both of New York, June 7.

GEORGE WILLIS BASS, Minneapolis, to Mrs. Frank E. Hills of New Hampshire, May 18.

OTIS HUNTER JONES to Mrs. Nancy Hovis Nichols, both of Charlotte, N. C., May 3.

JAMES LOUDEN BORLAND to Miss Margaret Gaillard, both of Jacksonville, Fla., May 1.

FRED W. OGDEN, Boston, to Miss Clara May Buchanan of New Orleans, June 17.

JOHN W. OVITZ JR., Sycamore, Ill., to Miss Virginia Noyes of Evanston, June 7.

JOHN RALPH RICE to Miss Helen Norine Ward, both of Elkton, Ky., May 15.

MCCHORD WILLIAMS to Miss Helen Louise Avery, both of Boston, May 17.

Deaths

Thomas Aloysius O'Brien ✠ Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1905; member of the American Academy of Ophthalmology and Otolaryngology; fellow of the American College of Surgeons; ophthalmic surgeon, St. Joseph's, Philadelphia General and Wills hospitals, Philadelphia, and the Fitzgerald-Mercy Hospital, Darby; aged 58; died June 21, at his summer home in Ship Bottom, N. J., of carcinoma of the right breast and fracture of a femur.

John Joseph Shaw ✠ Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1908; secretary of the state department of health; in 1940 received the Strittmatter award of the Philadelphia County Medical Society for his service as health officer; aged 57; died, June 24, in Harrisburg, Pa., of coronary occlusion.

George Loewenthal, Rhinebeck, N. Y.; Friedrich-Wilhelms-Universität Medizinische Fakultät, Berlin, Prussia, Germany, 1923; member of the Medical Society of the State of New York; on the staff of the Northern Dutchess Health Service Center; aged 41; died, June 23, of injuries received in an automobile accident.

Rastus Ransom Norris ✠ Crisfield, Md.; University of Maryland School of Medicine, Baltimore, 1904; served during the World War; formerly acting assistant surgeon, United States Public Health Service; aged 59; on the staff of the Edward W. McCready Memorial Hospital, where he died, June 9, of angina pectoris.

Howard Paul Benjamin, Omaha; John A. Creighton Medical College, Omaha, 1909; served with the British Army during the World War; aged 55; died, April 18, in the Veterans Administration Facility, Lincoln, of cirrhosis of the liver with portal obstruction and edema of the lungs.

Louis Edward Eslick, Rockwell City, Iowa; Eclectic Medical Institute, Cincinnati, 1894; member of the Iowa State Medical Society; served during the World War; past president of the Calhoun County Medical Society; formerly mayor; aged 72; died, May 28, of cerebral hemorrhage.

Duncan D. McArthur, Los Angeles; University of Southern California College of Medicine, Los Angeles, 1905; member of the California Medical Association; aged 66; died, May 30, in the Good Samaritan Hospital of coronary sclerosis and acute pulmonary edema.

Charles Vincent O'Brien, Brooklyn; Syracuse University College of Medicine, 1909; a staff physician at the Brooklyn College; aged 55; on the staffs of the Victory Memorial Hospital and St. Mary's Hospital, where he died, June 18, of cerebral hemorrhage.

Robert Goldsborough Owen ✠ Detroit; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1908; member of the American Society of Clinical Pathologists; served during the World War; aged 59; died, June 2, in the Harper Hospital.

Philip Gillett Cole ✠ New York; Columbia University College of Physicians and Surgeons, New York, 1910; served during the World War; director of the Tarrytown (N. Y.) Hospital; aged 57; died, June 30, of cerebral thrombosis.

Charles Fred Freytag, Los Angeles; Rush Medical College, Chicago, 1905; member of the California Medical Association; aged 59; died, June 12, of an incised wound of the throat, self inflicted with a razor blade.

Llewellyn George Petch, San Francisco; Cooper Medical College, San Francisco, 1904; served during the World War; on the staff of the United States Marine Hospital; aged 59; died, June 4, of cerebral hemorrhage.

George Paul Norton, Fitchburg, Mass.; Bellevue Hospital Medical College, New York, 1895; member of the Massachusetts Medical Society; aged 67; died, June 12, in the Burbank Hospital of intestinal obstruction.

Frank D. Crigler, Ludlow, Ky.; Medical College of Ohio, Cincinnati, 1884; aged 82; died, June 13, in the Good Samaritan Hospital, Cincinnati, of cholecystitis, postoperative peritonitis, arteriosclerosis and myocarditis.

Francis John Diamond, Ravenna, Mich.; Loyola University School of Medicine, Chicago, 1928; member of the Michigan State Medical Society; served during the World War; aged 42; died, June 21.

Gordon Ezra Woodruff, Louisville, Ky.; University of Louisville School of Medicine, 1937; captain in the British Army Medical Corps; aged 29; was killed recently near Accra, West Africa, while in action.

William Clark Tilden, Stanwood, Iowa; State University of Iowa College of Medicine, Iowa City, 1902; for many years member of the school board; aged 70; died, June 4, of cerebral hemorrhage.

John William Dixon ✠ Edgewood, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1897; fellow of the American College of Surgeons; aged 69; died, June 12.

Thomas Abraham Dean, Casper, Wyo.; Louisville (Ky.) Medical College, 1893; member of the Wyoming State Medical Society; aged 88; died in June in the Memorial Hospital.

David Thomson, Peterborough, Ont., Canada; Trinity Medical College, Toronto, 1894; L.R.C.S., L.R.C.P., Edinburgh, and L.R.F.P.S., Glasgow, 1895; aged 71; died, May 18.

Robert Thomas Poling, Columbus, Ohio; Ohio State University College of Medicine, Columbus, 1925; aged 41; resident, Franklin County Sanatorium, where he died, June 8.

George Frederick Zachritz, Faulkton, S. D.; St. Louis University School of Medicine, 1916; served during the World War; county health officer; aged 46; died, June 1.

Walter Herman Rietz ✠ Peoria, Ill.; Rush Medical College, Chicago, 1915; aged 51; died, June 5, in St. Francis Hospital of carcinoma of the rectum.

Charles Sanford Porter, Los Angeles; University of the City of New York Medical Department, New York, 1893; aged 79; died, April 21, of uremia.

Alfredo Russo, Providence, R. I.; Regia Università di Napoli Facoltà di Medicina e Chirurgia, Italy, 1900; aged 69; died, April 4, of myocarditis.

John O. Mathews, Memphis, Tenn.; Kentucky School of Medicine, Louisville, 1886; aged 83; died, June 9, at Dallas, Texas, of pulmonary abscess.

Petros Gerardus Hendricus Vander Wyst ✠ Altoona, Kan.; Hering Medical College, Chicago, 1912; aged 64; died, May 29, of heart disease.

Melvin Collins, Oxford, Kan.; Rush Medical College, Chicago, 1891; member of the Kansas Medical Society; aged 80; died, June 9, in Newton.

Isaac Smith Putnam, Dillonvale, Ohio; Starling-Ohio Medical College, Columbus, 1911; served during the World War; aged 52; died, June 5.

Samuel Mazor ✠ Philadelphia; Medico-chirurgical College of Philadelphia, 1914; aged 50; died, April 28, in the Temple University Hospital.

James M. Holtz, Etna, Pa.; Columbus Medical College, 1880; aged 93; died, June 1, in Alliance, Ohio, of myocarditis and arteriosclerosis.

Aaron J. Fulton, Blaine, Maine; University of Vermont College of Medicine, Burlington, 1890; aged 90; died April 21, of arteriosclerosis.

William Everett Kernan, Boston; Harvard Medical School, Boston, 1896; aged 68; died, June 4, of arteriosclerosis and heart disease.

Shuler Hardin Etheredge ✠ Tampa, Fla.; Medical College of the State of South Carolina, Charleston, 1924; aged 41; died, June 10.

Douglas C. Ramsey, New Providence, N. J.; St. Louis Medical College, 1880; aged 81; died, May 22, of chronic myocarditis.

Charles Wesley House, Auburn, N. Y.; Eclectic Medical Institute, Cincinnati, 1886; aged 78; died, June 2, of cerebral hemorrhage.

George R. Frey, Milwaukee; Milwaukee Medical College, 1906; aged 72; died, June 29, of acute miliary tuberculosis and myocarditis.

Allen Monroe Ottman ✠ Rochester, N. Y.; Albany (N. Y.) Medical College, 1897; aged 66; died, June 20, of coronary thrombosis.

Thomas R. Mullen, Omaha; John A. Creighton Medical College, Omaha, 1896; aged 73; died, April 29, of Parkinson's disease.

Frank Elbert Tolle ✠ Overland Park, Kan.; University of Kansas School of Medicine, Kansas City, 1932; aged 33; died, May 3.

Bureau of Investigation

FERNEL PROMOTES LANDA, A FRAUDULENT BUST DEVELOPER

The Postmaster General has issued a fraud order against Landa, Beauty-Health Institute and their officers and agents as such at Chicago. Dr. Jean Paul Fernel was engaged in the advertisement and sale through the mails of preparations called "Landa" and "Melanine" as treatments for the development of the female breast and for the restoration of natural color to gray hair respectively.

The directions for using the first of these preparations, Landa, stated "Take two tablets after each meal three times daily." Chemical analysis indicated that the tablets contained zinc phosphide, calcium sulfate, siliceous material and had an iodine content of 0.033 per cent. There was also an indication of the presence of damiana and of some other plant material which the promoter claimed was "European goat's rue."

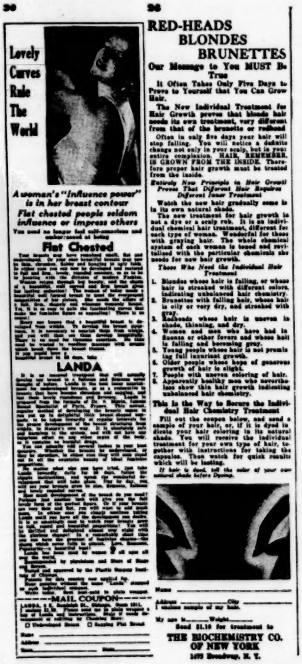
Expert medical evidence was introduced to show the various factors concerned in the development of the human breast and that the ingredients contained in the remedy would not affect the size or the shape of the breast. It was pointed out that the taking of the preparation Landa will not and cannot restore diseased ovaries or pituitary glands to normal function. The evidence before the solicitor showed that the use of Landa as directed will not and cannot produce in "flat chested women" a "steady, firm, permanent growth" of the breasts to any size desired as represented by the promoters of this scheme; furthermore, that the use of the preparation mentioned will have no effect in restoring female breasts which have "lost their lovely contour" to "lovely curves" as represented. It was pointed out that the claims made that this preparation will enlarge and beautify the female breasts and that it will restore lovely contours to those lacking therein were shown by medical evidence to be false and fraudulent.

The other preparation mentioned in this order was recommended to be used as follows: "Take one capsule before meals three times daily and at bed-time, followed by a glass of water, or fruit juice or vegetable juice." Chemical analysis indicated that this preparation contained, essentially, powdered seaweed (0.02 per cent iodine).

Evidence was brought forth that gray hair is due to many factors, most of them concomitant of age, and that iodine has no recognized value in the treatment of the conditions which may cause the hair to become gray and, therefore, that the representations that Melanine will restore gray hair to normal color results in the defrauding of persons of amounts remitted therefor.

It was noted in the fraud order that Dr. Jean Paul Fernel, the promoter of this enterprise, referred to his medical graduation and to the fact that he had practiced in Chicago but that he made no defense whatsoever of the claims made for the preparations and that, therefore, the promotion of these products constituted a scheme for obtaining money through the mails by means of false and fraudulent pretenses, representations and promises.

An advertisement for Landa, which appeared in a publication for which Fernel was consultant, is reproduced herewith.


Lovely Curves Rule The World
A woman's "influence power" is in her breast contour
Flat chested people seldom influence or impress others
Flat Chested
LANDA
RED-HEADS
BLONDES
BRUNETTES
Our Message to You MUST Be True
It Often Takes Only Five Days to Prove to Yourself that You Can Grow Hair.
The New Individual Treatment for Hair Growth proves that blonde hair needs its own treatment, very different from that of the brunette or redhead
Often in only five days your hair will stop falling. You will notice a definite change not only in your scalp, but in the entire complexion. HAIR REMEMBERS. IS GROWN FROM THE ROOTS. Therefore proper hair growth must be treated from the inside.
Entirely New Principle in Hair Growth Proves That Different Hair Calls for Different Inner Treatment
Watch the new hair growth come in in its own natural shade.
The new treatment for hair growth is not a dye or a scalp rub. It is a new, different, individual method, different for each type of blonde. Wonderful for those with graying hair. The whole chemical system of each woman is tested and then tailored with the particular chemicals she needs for new hair growth.
Those Who Need the Individual Hair Treatment
1. Blondes whose hair is falling, or whose hair is streaked with different colors, indicating unbalanced hair chemistry.
2. Brunettes with falling hair, whose hair is oily or very dry, and streaked with gray.
3. Redheads whose hair is uneven in shade, thinning, and dry.
4. Women and men who have had in fevers or other fevers and whose hair is falling and becoming gray.
5. Young people whose hair is not proving the full luxuriant growth.
6. Older people whose hope of generous growth of hair is almost gone.
7. Parents of girls in adolescent period of hair.
8. Apparently healthy men who nevertheless show thin hair growth indicating unbalanced hair chemistry.
This is the Way to Secure the Individual Hair Chemistry Treatment
Fill out the coupon below, and send a sample of your hair, or, if it is short, pluck your hair entering in its natural shade. We will send you the right treatment for your own type of hair, together with instructions for taking the capsules. Then watch for quick results which will be lasting.
Just a few, if dyed, tell the color of your own natural shade before Dyeing.
Name
Address City
I enclose sample of my hair.
My age is Weight
Send $3.50 for treatment to
THE BIOCHEMISTRY CO. OF NEW YORK
1472 Broadway, N. Y.
MAIL COUPON

Advertisements from *Slimming and Gaining*, Winter Number 1936-37. Edited by Edna Purdy Walsh. Jean Paul Fernel, M.D., Consultant.

THE BIOCHEMISTRY COMPANY

Mrs. Edna Purdy Walsh Fernel President of Firm Promoting a Fraudulent Hair Grower

The Post Office Department has issued a fraud order against Biochemistry Publishers, Biochemistry Publishers, Inc., Vitamin Garden Products and Scientific Eating, at Freeport, Ill.; Biochemistry Co., High Intensity Foods and Organo Chemistry Co., at New York; Preventive Food Elements, at Battle Creek, Mich., and Nurse Oliphant Co., at Peoria, Ill., and their officers and agents as such, for the promotion through the mails of a preparation called "Internal Hair Treatment" which was represented as being capable of quickly and permanently growing hair on the heads of all users and another item called "Spermadrine" which was represented as being efficacious in the prevention and cure of nervous breakdowns, sex failure and prostate trouble, with the claim that it contained no drugs.

It was further charged that other preparations, known as calcium, silicon, manganese, iodine, iron, chlorine, fluorine, sodium and phosphorus, were sold under fraudulent representations to the effect that they will cure or prevent anemia, falling hair, poor eyesight, "muddy complexion," insanity, lost vitality, obesity, arthritis, catarrh, deafness, cataracts, ulcers and tuberculosis. In addition a so-called "night brassiere" was falsely represented as a means of restoring all "sagging or flabby and overdeveloped" female breasts to "tone and beauty."

It was stated in the memorandum that the officers of this incorporated business were Mrs. Edna Purdy Walsh Fernel, President, and Mr. Orville S. Thompson, Vice President, Secretary-Treasurer and office manager.

The internal hair treatment was said to consist of powdered seaweed (iodine 0.009 per cent), animal glandular material and zinc phosphate. Evidence was introduced to show that none of the ingredients in the treatment would have any effect whatsoever in the prevention or elimination of the conditions mentioned, that the preparation will not cause hair to grow and that the item is worthless in the treatment of any condition causing loss of hair. (See advertisement reproduced herewith.)

The second product mentioned specifically in this order was Spermadrine which was promoted for sex failure. *Chemical analysis was found to be the same as that for Internal Hair Treatment.* Medical evidence adduced at the hearing showed that the preparation would be of no value in the prevention or cure of any of the causes of the conditions for which it is promoted, and that the claim that Spermadrine was not a drug was also false.

The chemicals were advertised with specific claims for each. The Post Office Department stated that they will not prevent or cure the conditions and diseases for which they were sold.

The "night brassiere" was fitted with sponges which when moistened gave off an extract consisting of iodine, sodium chloride, calcium, magnesium, iron, sulfur and other ingredients of seawater. Testimony indicated that amounts of the minerals given off are not absorbed and cannot have any effect on the conditions for the treatment of which the device was sold.

It was stated that no persons with any medical training were connected with the operation of the business, and it was declared to be a scheme for obtaining money through the mails by means of false and fraudulent pretenses, representations and promises.

HUMAN RELATIONS, INC., VITAMINE FARM OF GENEVA, ETC.

Peter Furno and Others Mentioned in Fraud Order Involving Various Products

The Post Office Department has issued a fraud order against Human Relations, Inc., Nurse Chalmers Co., Ann Phillips Co., Intensified Food Products, Diet Revitalization, Health Information Bureau, Vitamine Farm of Geneva, Lora Holm, Manager, Holland Food Chemicals, Ocean Dew Gardens, Healing Magazine, Defensive Foods Co. and their officers and agents as such, at Chicago. Under the names mentioned in the order, the memorandum states that Messrs. Allen Block, L. A. Neis and Peter Furno were engaged in the advertisement and sale through the mails of a number of preparations and a device for the treatment of various diseases and conditions. One of these products was known as "Vagilaxis" or "Herb Spheres No. 3," which was recommended for the treatment of vagilaxis and which, on chemical analysis, was shown to consist of 4.55 per cent boric acid, 0.16 per cent copper sulfate, 0.36 per cent alum, combined in a base of glycerin and gelatin and made into large bottle shaped greenish suppositories. The directions for use of this preparation were as follows: "The parts should be well cleansed with douche of warm water and a suppository inserted on retiring."

Medical evidence showed that the only effect from the use of such a preparation would be a temporary shrinking of the superficial membranes, which is of no practical or lasting value in the treatment of relaxed vaginal walls. Medical evidence further showed that this preparation when used as directed will not and cannot restore "stretched, flabby" "body tissues" and "mucous membranes" of women users to their normal "tone," "elasticity" and "resilience," as represented in the advertisement of this preparation, and that, therefore, the claims to that effect were false and fraudulent.

The second item mentioned in this order was promoted under the name "Ann Phillips Carrot Oil," which was shown to contain a potency of 2,820 vitamin A units per gram and was recommended as a "wonderful inner dermatological treatment for acne" and for "muddy, pale skin of greasy character," with the following directions: "5 drops 3 times daily, before meals with lemon juice."

Expert testimony was introduced to show that there is no scientific evidence that the lack of vitamin A is responsible for color changes in the skin or other factors that involve the complexion and that such characteristics as muddiness, greasiness, pallor and other defects are caused by many diseases not connected with vitamin A deficiency. It was contended, therefore, that the use of this "Carrot Oil" will not and cannot restore a healthy complexion to all persons suffering from the conditions mentioned, and the solicitor found from this evidence that such representations were false and fraudulent.

The third item mentioned in this fraud order was promoted as "Van Hagen's Brain Food," which was found to consist essentially of lecithin, cerebrin, cholesterin, fats, phospholipins, glycerin, phospholglyceric acid, alfalfa and almond extract. The principal ingredients in this preparation constitute the component parts of egg yolk. The directions were as follows: "Take one tablespoonful twice daily, midway between meals. Shake well before pouring." The preparation was recommended for rebuilding nerve cells.

Medical evidence showed that it would not rebuild nerve cells or correct dysfunctions of "sight, speech, hearing and gland potency" as represented, nor will the preparation restore physical energy in cases in which that quality is lacking and, therefore, that such representations are false and fraudulent.

The fourth item mentioned was known as "Mineral Mix Capsules." These were represented to contain calcium, silicon, fluorine, sulfur, iron, manganese, oxygen, spleen, pectin, potassium, sodium, chlorin, iodine, cholagogues, enzymes and gelactans. Some of these elements were mixed together and sold for catarrh, deafness and arthritis, others for colitis and still other mixtures for liver and gallbladder diseases. The Post Office memorandum noted that such conditions are due to many various factors, necessitating for their proper treatment individual care in each instance, and that some of the conditions are of a very serious nature and are incurable. The evidence showed that the use of the preparations will not and cannot cure persons suffering from these diseases and, therefore, that the representations made for the preparations are false and fraudulent.

The fifth item mentioned in this order is a device known as the "Sleeping Brassiere." Examination disclosed that it contained small rubber sponges to be worn over the breast, which were to be moistened with water. The addition of water results in the device giving off an extract containing sodium chloride, calcium, magnesium, iron, sulfur and a very small amount of iodine. Evidence was introduced to show that very little of the ingredients mentioned were absorbed when the device was worn in the manner indicated. Medical evidence adduced indicated that the wearing of the device as directed will not and cannot, according to the medical evidence, reduce the size of overdeveloped busts. Neither will its wearing firm the breast as claimed, nor will it have any permanent or real effect on the shape, size or consistency of the breast. The solicitor found from this evidence that the representations employed in the sale of this device were false and fraudulent.

YOU CAN HAVE A "DRAG" WITH NATURE

ARE YOU GOING AROUND CHEMICALLY NUDE?

Clothe yourself inwardly with the 12 white magic elements, and you will become Nature's Darling

Read About That Missing "Something" in the Woman and Man of Today

It very often happens that the smallest things are the greatest in power. The twelve mineral elements of the body for instance, are present in only very small amounts, yet each one of them is a great General managing the bulk of the body. When one of these minerals is low, all of the others sometimes suffer. When one is completely gone, life continues probably on some other planet, but not here on earth.

When Phosphorus, Silicon and Calcium are low for instance, we call the person tubercular. When Fluorin is low find infectious, poor endurance, erratic personalities, bone disease, premature age.

When Potassium is low we lack "come-back" pep, enthusiasm, and healing power. We have little elasticity of body or mind. We are constipated, sometimes morbid, and also, when we lack Phosphorus and Potassium, pain is greatly exaggerated.

When Sodium and Chlorin are low, we are catarrhal, suffer with stiff joints, deafness, acidity, and many distressing diseases. We need Sodium and Chlorin for alkalinity, spontaneity, cleansing of the cells. Sodium also keeps us from stiffening processes in bones and arteries by keeping Calcium in solution.

The nervous fearful person needs Iodine, Phosphorus, Manganese, Magnesium, and often Sulphur. All elements are needed, however, by the nervous, low in vitality. They need with the above elements, Calcium and Silicon to give greater endurance, as well as Sodium and Potassium to give muscular development and nourishment.

Anemic people need Iron, Iodine, Phosphorus and Calcium. Diabetics are hungry for Sodium. Those with failing hair need Silicon, Fluorin and Sulphur, as well as Potassium.

These few facts are only a few of the truths regarding the twelve master chemicals of the body. We all need all of these minerals, but some of them are given to great advantage in groups, or alone.

Sickness comes from both wrong thinking and wrong eating. The God-given elements are put in the earth to aid our bodies to become more divine instruments. When we lack one of the 16 chemical elements which make this body, we are rejecting God-given power, and w.. fail ill, or pass out of this physical frame. We are nervous, tired, forgetful perhaps at first. Then other things follow, with grave consequences.

Read of what the twelve minerals do for you. How they regulate your thought, give you new purpose, change your appearance. Most of us who work mentally are deficient in these elements. They are small, but how powerful, you will only know when you make them a part of your body. Let them aid you in your mental healing, so that you can quickly be the person you want to be.

Every one of these chemicals is derived from foods and foods alone. Drugless medicines of great power they are. They are absolutely pure, absolutely clean, and everyone is grown under our supervision. No preservatives or adulterants of any kind are added. The foods are dehydrated, powdered and put into dainty capsules so that no matter where you are you may have your twelve life giving chemicals.

Each order is prepared individually, and fresh, at the time of the order. No wholesale stocks to deteriorate. Order your foods five days in advance of your needs, allowing us time to prepare your individual order. Our foods are prepared with loving care. The vibration of 100 percent wishes for your health is put into every little capsule.

"You love every one of these capsules, don't you," said a visitor to the Vitamine Farm. "The whole place reflects it."

Note—Capsules being foods, never interfere with each other, all may be taken together if desired.

"I feel like Nature's special pet since I learned about vitamines and elements.

THOSE TWELVE MINERAL SALTS WHICH KEEP YOU ALIVE WHEN YOU LIVE

THE VITAMINE FARM of Geneva

Send all orders to Chicago Office: 360 N. MICHIGAN AVE., CHICAGO, ILL.

Advertisement from *Slimming and Gaining*, Winter Number 1936-37. Edited by Edna Furdy Walsh. Jean Paul Fernel, M.D., Consultant.

Correspondence

AUSCULTATORY PERCUSSION AS A DIAGNOSTIC METHOD

To the Editor:—When auscultatory percussion is used as a routine it will be found to be accurate for locating the apex of the heart. It is also of advantage in examination of the lungs. The simplicity of the method and its ready adaptability plus its assured accuracy should warrant its wide usage, especially by the keen diagnostician.

To locate the apex of the heart the stethoscope is held on the manubrium sterni, and light percussion is made beginning at the midaxillary line in the fifth left interspace. A definite change in sound will be detected as soon as the percussion reaches the left border of the heart. After the border of the heart is ascertained in each interspace, the distance to the right and left of the midsternal line may be recorded in centimeters.

Auscultatory percussion is of advantage also in examining the upper part of the chest anteriorly and posteriorly to detect minor changes in the apexes. For examination of the clavicular and subclavicular areas the stethoscope is placed over the upper dorsal vertebrae and light percussion is made with the finger over the clavicles and subclavicular areas. For examination of the posterior portion of the chest the stethoscope is placed over the manubrium sterni and light percussion is made over the upper part of the back. The method has proved a valuable diagnostic aid, especially if one uses the disk stethoscope. The examiner stands on the right side of the patient when examining the anterior part of the chest and on the left side of the patient when examining the upper posterior areas of the chest if the examiner is right handed, or vice versa if left handed.

My attention was first drawn to this method of outlining the heart by Dr. Henry Suydam Satterlee while we were on service at the Willard Parker Hospital in 1914. Dr. Satterlee used a light percussion hammer made of a 10 inch length of glass tubing 3/16 inch in diameter. The tubing is bent at right angles to form a hammer head 2 inches long. In the handle portion a centimeter scale 19 cm. long is introduced. The ends of the tube are sealed with sealing wax. The percussion end is partly covered with rubber tubing.

Percussion with the finger may, of course, be substituted for the glass percussion hammer. Friction with a toothpick, an applicator or a vibrating tuning fork may be used similarly.

C. T. Sharpe, M.D., New York.

MEDIASTINOCARDIAC REACTION AFTER IRRADIATION

To the Editor:—In The Journal, June 14, page 2648, Dr. Edward Rose and Dr. Charles C. Wolferth report an acute mediastinocardiac reaction following irradiation for hyperthyroidism and describe 3 such cases, 2 occurring in 1937 and 1 in 1940. We have just reviewed 58 cases of hyperthyroidism treated in our service at Bellevue Hospital during the past fifteen years and in no instance have we seen any complication such as mediastinocardiac reaction. These unusual reactions reported by Rose and Wolferth are interesting, but I do not believe that their interpretations warrant the statement that irradiation was the cause. Were this phenomenon a frequent occurrence, the profession should have heard about it sooner. It seems peculiar that cases occurring in 1937 should be reported only now. In my opinion other causative factors are responsible for the condition reported by these authors.

Ira I. Kaplan, M.D., New York.
Director, Radiation Therapy, Bellevue Hospital.

PINWORM INFECTION IN A FAMILY

To the Editor:—Relative to my article entitled "Acute Appendicitis and Pinworm Infection Occurring Concomittantly in the Same Family" (The Journal, Sept. 21, 1940, p. 1009), I have obtained more information concerning this family during a visit to the Warren General Hospital in Warren, Pa.

The mother, listed as Mrs. T. in the article, was admitted to the Warren General Hospital on April 2, 1941 with the complaint of pain in the lower part of the abdomen, more particularly in the right lower quadrant. There was no associated diarrhea or vomiting. Because of persistence of pain, an appendectomy was performed on April 4, and a slightly injected appendix, with its tip bound down, was found and removed. When the appendix was opened, actively motile pinworms were found present.

The blood counts taken preoperatively showed 2 per cent eosinophils. The postoperative course was uneventful.

Victor Mayer, M.D., New York.

HAZARD OF DIESEL ENGINE OIL UNDER PRESSURE

To the Editor:—In The Journal, June 28, page 2848, is an article on the injury of a hand with subsequent loss of fingers due to jets of Diesel engine oil impinging on the skin at high pressure.

In 1935 Dr. John F. Roberts presented a thesis for the degree of doctor of medical science on infiltration of tissues with medicaments at high pressure. That is not the exact title of the thesis, but it gives the sense.

Some time earlier Mr. Arnold Sutermeister, a mechanical engineer in Brooklyn, had a man working for him whose hand was hit by a very fine jet from a defect in a copper pipe carrying Diesel oil at high pressure. There was no immediate pain, and no wound could be seen. The man wiped off the oil and went on with his work. The next day the hand pained and was lanced by a physician. I think it was the thumb. A small collection of oil was found beneath the periosteum next to the bone. This was evacuated and recovery was uneventful.

In the case reported there is the possibility that the oil might have been found and evacuated had an immediate incision been made. Unfortunately Dr. Roberts' thesis, which is rather long, has never been printed but is on deposit in our library.

Mr. Sutermeister tried directing a jet of water at 9,000 pounds per square inch on the palm of his own hand and could see no effect, but on looking at the back of the hand he saw a "water blister." He brought the matter to the attention of our department of surgery, and Dr. Roberts worked on it as a means of painlessly injecting local anesthetics. Later a more powerful and convenient device than the one he used was developed in an experimental form under my guidance, but when tested on a guinea pig it was found to be capable of doing serious damage to tissues. It is now in the hands of Dr. J. C. Fiala at Memorial Hospital. He was interested in the possibility of using it in the treatment of deep tumors.

The track of the liquid when seen under a microscope is definite but very small. Many sheets of paper can be penetrated and tin or lead foil can be penetrated and, by moving the jet, can be cut. Dr. Hughes's paper is timely, and I am glad that he has brought the matter to the attention of the profession.

Horatio B. Williams, M.D.,
Columbia University,
New York.

Medical Examinations and Licensure

COMING EXAMINATIONS AND MEETINGS

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE

CHICAGO, Feb. 16-17, 1942. Council on Medical Education and Hospitals, Sec., Dr. William D. Cutter, 535 North Dearborn Street, Chicago.

NATIONAL BOARD OF MEDICAL EXAMINERS EXAMINING BOARDS IN SPECIALTIES

Examinations of the National Board of Medical Examiners and Examining Boards in Specialties were published in THE JOURNAL, July 26, page 313.

BOARDS OF MEDICAL EXAMINERS

ALABAMA: Montgomery, June 16-18. Sec., Dr. J. N. Baker, 519 Dexter Ave., Montgomery.

ARKANSAS: * *Medical.* Little Rock, Nov. 6-7. Sec., Dr. D. L. Owens, Harrison. *Eclectic.* Little Rock, Nov. 6. Sec., Dr. Clarence H. Young, 1415 Main St., Little Rock.

CALIFORNIA: *Oral examination* (required when reciprocity application is based on a state certificate or license issued ten or more years before filing application in California), San Francisco, Oct. 1. *Written.* Sacramento, Oct. 20-23. Sec., Dr. Charles B. Pinkham, 1020 N St., Sacramento.

GEORGIA: Atlanta, Oct. 14-15. Sec., State Examining Boards, Mr. R. C. Coleman, 111 State Capitol, Atlanta.

IDAHO: Boise, Oct. 7. Dir., Bureau of Occupational License, Mr. Walter Curtis, 355 State Capitol Bldg., Boise.

ILLINOIS: Chicago, Oct. 14-16. Supt. of Registration, Mr. Lucien A. File, Department of Registration and Education, Springfield.

INDIANA: Indianapolis, June 16-18. Sec., Board of Registration and Examination, Dr. J. W. Bowers, 301 State House, Indianapolis.

KANSAS: Topeka, Sept. 23-24. Sec., Board of Medical Registration and Examination, Dr. J. F. Hassig, 905 N. 7th St., Kansas City.

MARYLAND: *Medical.* Baltimore, Dec. 9-12. Sec., Dr. John T. O'Mara, 1215 Cathedral St., Baltimore. *Homeopathic.* Baltimore, Dec. 9-10. Sec., Dr. John A. Evans, 612 W. 40th St., Baltimore.

MICHIGAN: * Lansing, Oct. 15-17. Sec., Board of Registration in Medicine, Dr. J. Earl McIntyre, 203 Hollister Bldg., Lansing.

MINNESOTA: * Minneapolis, Oct. 21-23. Sec., Dr. J. F. Du Bois, 230 Lowry Medical Arts Bldg., St. Paul.

MISSISSIPPI: *Reciprocity.* Jackson, December. Asst. Sec., State Board of Health, Dr. R. N. Whitfield, Jackson.

MONTANA: Helena, Oct. 6-8. Sec., Dr. Otto G. Klein, First National Bank Bldg., Helena.

NEVADA: *Reciprocity with oral examination,* Aug. 4. Sec., Dr. Fred M. Anderson, 215 N. Carson St., Carson City.

NEW HAMPSHIRE: Concord, Sept. 11-12. Sec., Board of Registration in Medicine, Dr. T. P. Burroughs, State House, Concord.

NEW JERSEY: Trenton, Oct. 21-22. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO: Santa Fe, Oct. 13-14. Sec., Dr. Le Grand Ward, 135 Sena Plaza, Santa Fe.

NEW YORK: Albany, Buffalo, New York and Syracuse, Sept. 15-18. Chief, Bureau of Professional Examinations, Mr. Herbert J. Hamilton, State Education Department, 315 Education Bldg., Albany.

VERMONT: Burlington, Feb. 10-12. Sec., Board of Medical Registration, Dr. F. J. Lawliss, Richford.

VIRGINIA: Richmond, Dec. 9-12. Sec., Dr. J. W. Preston, 30½ Franklin Road, Roanoke.

WASHINGTON: * Seattle, July 21-23. Sec., Department of Licenses, Mr. Nelson N. Vaughan, Olympia.

WISCONSIN: * Madison, Jan. 13-15. Sec., Dr. H. W. Shutter, 425 E. Wisconsin Ave., Milwaukee.

WYOMING: Cheyenne, Oct. 6-7. Sec., Dr. M. C. Keith, State Capitol Bldg., Cheyenne.

* Basic Science Certificate required.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ARIZONA: Tucson, Sept. 16. Sec., Mr. Franklin E. Roach, Science Hall, University of Arizona, Tucson.

COLORADO: Denver, Sept. 10-11. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver.

CONNECTICUT: Oct. 11. Address State Board of Healing Arts, 1945 Yale Station, New Haven.

DISTRICT OF COLUMBIA: Washington, Oct. 20-21. Sec., Dr. George C. Ruhland, 203 District Bldg., Washington.

FLORIDA: DeLand, Nov. 1. Sec., Professor J. F. Conn., John B. Stetson University, DeLand.

MINNESOTA: Minneapolis, Oct. 7-8. Sec., Dr. J. C. McKinley, 126 Millard Hall, University of Minnesota, Minneapolis.

NEBRASKA: Lincoln, Oct. 7-8. Dir., Bureau of Examining Boards, Mrs. Jeannette Crawford, 1009 State Capitol Bldg., Lincoln.

OREGON: Portland, Nov. 1. Sec., State Board of Higher Education, Mr. Charles D. Byrne, University of Oregon, Eugene.

RHODE ISLAND: Providence, Aug. 20. Chief, Division of Examiners, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

SOUTH DAKOTA: Aberdeen, Dec. 5-6. Sec., Dr. G. M. Evans, Yankton.

WISCONSIN: Madison, Sept. 20. Sec., Professor Robert N. Bauer, 3414 W. Wisconsin Ave., Milwaukee.

Wyoming Endorsement Report

The State of Wyoming Board of Medical Examiners reports 5 physicians licensed to practice medicine by endorsement on February 3. The following schools were represented:

School	LICENSED BY ENDORSEMENT	Year Grad.	Endorsement of
University of Arkansas School of Medicine		(1926)	Arkansas
University of Nebraska College of Medicine (1935), (1936)		(1924),	Nebraska
Vanderbilt University School of Medicine		(1934)	Tennessee

Tennessee March Report

The Tennessee State Board of Medical Examiners reports the written examination for medical licensure held at Memphis, March 26-27, 1941. The examination covered 10 subjects and included 100 questions. An average of 75 per cent was required to pass. Twenty-six candidates were examined, all of whom passed. The following schools were represented:

School	PASSED	Year Grad.	Per Cent
University of Tennessee College of Medicine 81, 84.2, 84.6, 85, 85.1, 85.6, 85.8, 86.6, 86.6, 86.7, 87.2, 87.6, 87.6, 87.6, 87.7, 87.7, 87.7, 88.2, 88.8, 88.9, 89.2, 91.2		(1941)	80.6,
University of Wisconsin Medical School		(1928)	89.1
University of Toronto Faculty of Medicine		(1938)	87.2
McGill University Faculty of Medicine		(1940)	84.4

Rhode Island April Report

The Rhode Island Board of Examiners in Medicine reports the written examination for medical licensure held at Providence, April 3-4, 1941. The examination covered 8 subjects and included 50 questions. An average of 80 per cent was required to pass. Four candidates were examined, all of whom passed. The following schools were represented:

School	PASSED	Year Grad.	Per Cent
Boston University School of Medicine		(1939)	86.6
Harvard Medical School		(1938)	80
Tufts College Medical School		(1940)	84.1
Jefferson Medical College of Philadelphia		(1939)	86.6

Bureau of Legal Medicine and Legislation

MEDICOLEGAL ABSTRACTS

Dental Practice Acts: Failure of Board in Revocation Proceedings to Grant Bill of Particulars.—In revocation proceedings instituted by the Arizona State Dental Board Davis, a licensed dentist, was charged with employing a named unlicensed person to make dentures, an act that under the Arizona dental practice act can be performed only by a person licensed to practice dentistry. Davis appeared at the hearing on May 28, 1938 and moved for a bill of particulars to inform him of the names of the persons for whom the unlicensed person had allegedly performed services and the particular work done on each person on which the board would rely to sustain the charges. The motion was denied and the hearing proceeded. Witnesses testified with respect to dental work which the named unlicensed person did on them and of the connection of the accused dentist with such work so far as they were aware. The hearing was then continued to June 11. At that time the witnesses who had testified previously were recalled and were fully cross examined by counsel for the dentist, and the unlicensed person testified and was cross examined. The dentist then presented such evidence as he desired, and three witnesses, including the dentist himself, testified on his behalf. Thereafter the board revoked his license, which action was affirmed by the superior court, Maricopa County, in an action of certiorari subsequently brought by the dentist. The dentist then appealed to the Supreme Court of Arizona.

The principal point relied on by Davis on appeal was the failure of the board to grant him a bill of particulars. Had the hearing, said the Supreme Court, been concluded on May 28 without giving the dentist an opportunity to know before the hearing the names of the witnesses on whom the work by his unlicensed employee was alleged to have been done and the character of the work done on them, and the order of revocation made at that time, we believe that it would have been fatal to the jurisdiction of the board. Quasijudicial bodies, such as the state dental board, are not bound by all the methods of procedure used in the more formal trials provided for in court, and proceedings before them are not criminal and do not require that every doubt be resolved in favor of the innocence of the

licentiate. Nevertheless the fundamental principles of law and equity do apply to such proceedings and the accused must have knowledge of the charges made against him and a reasonable opportunity to meet them. A violation of this requirement goes to the jurisdiction of the body to act at all. To compel the dentist to go to trial without knowing, at such a time and in such a manner that he could properly prepare to refute their testimony, the names of the witnesses to be used against him and what violation of the dental act they would testify he had committed would have deprived him of a fundamental right. It was not necessary, however, that he should have been apprised of these facts at any particular time and in any particular manner, so long as he had a proper opportunity to make a defense. The record shows that the witnesses against him testified on May 28 and on that day he had full knowledge not only of what he was entitled to know, to wit, who they were and what the substance of their testimony would be, but what it actually was, and he was then given until June 11 to prepare his defense. Under these circumstances he had ample opportunity to meet their testimony and the failure to grant a bill of particulars when demanded did not go to the jurisdiction of the board.

The dentist contended further that the evidence before the board was insufficient to sustain the charges preferred against him. In a certiorari proceeding, said the Supreme Court, neither this court nor the trial court passes on the weight of the evidence. It is sufficient if there is testimony before the board that, if believed by a reasonable man, would show the jurisdiction of the board to act. The evidence before the board was ample in both quantity and quality, if believed by the board, to have justified it in finding that the dentist had violated the dental practice act, as charged, not once but repeatedly and continuously.

The dentist contended finally that the penalty of revocation was so severe that the Supreme Court might modify it. The dental practice act, said the Supreme Court, is explicit as to the only penalty which may be inflicted for a violation of it, namely the revocation of a license to practice dentistry. While the penalty is severe, it is not beyond the power of the legislature to inflict, and this court may not substitute its judgment for that of the legislature on this point.

The court accordingly affirmed the order of the board revoking Davis's license to practice.—*Davis v. Arizona State Dental Board, 112 P. (2d) 877 (Ariz., 1941).*

Medical Lien Act (Washington): Lien Applies Only with Respect to Services Rendered After Effective Date of Act.—Evelyn Maricelli was injured on Aug. 30, 1936 in an automobile accident occasioned by the negligence of the driver of the car in which she was riding. She was taken to a hospital conducted by the Columbia Clinic, Inc., and remained there, receiving treatment and care from physicians and other employees of the clinic, until Feb. 23, 1937. She executed a release and effected a settlement with the driver's insurer for $4,000 and court costs on Feb. 18, 1938, on which day the insurer delivered to her a draft for that sum payable jointly to her and to the clinic. The clinic refused to endorse the draft until it had been paid approximately $1,500, which it claimed for the hospital and medical services it had rendered and for the payment of which on the following day it for the first time filed a claim of lien by virtue of the Washington medical lien act (Laws, 1937, ch. 69). That act was enacted March 13, 1937, which was subsequent in time to the completion of all the services rendered by the clinic except two roentgenograms, which were taken in October and November 1937. Section 1 of the lien act, under which the clinic's claim of lien was asserted, provides in part, as follows:

> Every operator of a hospital and every duly licensed nurse, practitioner, physician and surgeon rendering service for any person who has received a traumatic injury shall have a lien upon any claim, right of action and/or money to which such person is entitled against any *tort feasor* and/or insurer of such *tort feasor* for the value of such service, together with costs and such reasonable attorney's fees as the court may allow, incurred in enforcing such lien: . . . *Provided, further,* That all the said liens for service rendered to any one person as a result of any one accident shall not exceed twenty-five (25) per centum of the amount of an award, verdict, report, decision, decree, judgment or settlement.

Miss Maricelli returned the draft to the insurer the day that the clinic refused to endorse it and demanded that she alone be paid in full in accordance with the terms of the settlement. The insurer, however, the following day, but before the clinic had filed its claim of lien, paid her $3,000 and court costs and retained one quarter of the settlement to protect itself against the possibility that the clinic would successfully assert a lien on one quarter of the settlement. Later Miss Maricelli assigned to her mother, the plaintiff in the present proceedings, her claim to the $1,000 retained by the insurer, and the plaintiff instituted an action against the insurer, to which the clinic and the driver were subsequently joined as parties. The insurer paid the $1,000 into the registry of the court and was dismissed from the action. The cause then proceeded against the clinic alone, and the trial court ordered that the money deposited with it be paid to the clinic. The plaintiff then appealed to the Supreme Court of Washington.

Section 2 of the lien act provides as follows:

> No person shall be entitled to the lien given by the preceding section unless he shall, within twenty (20) days after the date of such injury, *or, if settlement has not been a[e]ffected with and payment made to such injured person, then at any time before such settlement and payment,* file for record with the county auditor of the county in which said service was performed, a notice of claim stating [certain prescribed details].

The plaintiff contended first that the clinic had not timely asserted and filed its claim for lien. This contention was based on the argument that the release executed by Miss Maricelli on Feb. 18, 1938 and the acceptance by her at that time of the original draft constituted a settlement and payment which occurred before the clinic had filed on February 19 its claim of lien as required by statute. The court, however, did not agree with the contention. In the first place, said the court, the section of the statute quoted immediately above requires only that the notice of claim for lien be filed prior to the time that settlement is effected with, and payment made to, the injured person. Although the original draft was delivered to Miss Maricelli personally, it was not in settlement and payment of her claim alone but for that of the clinic as well, because the draft was made payable to the two parties jointly. The full amount of the draft was neither intended for, nor received by, her alone. The very theory of the plaintiff's action throughout has been that Miss Maricelli has not been paid the full amount of the settlement. Hence, so far at least as she had not received payment, the time for filing a lien had not expired. In the second place, if the original draft is considered as settlement with, and payment to, Miss Maricelli alone, she repudiated it by returning the draft, demanding and accepting another and agreeing to the conditions under which the second draft was issued. At that time, moreover, the clinic's claim for lien had not been filed. In the third place, so far as the $1,000 here involved is concerned, there had never been complete settlement and payment even as to Miss Maricelli. On the contrary, a portion of the total amount agreed on as a settlement was specifically withheld to await the judgment of the court in this action. Since the lien was filed prior to payment of the amount here in question to the injured person as contemplated by the statute, the filing was timely.

The plaintiff next contended, in effect, that the clinic actually had no lien, since the lien act cannot be construed retroactively and hence the lien provided therein applies only with respect to services rendered subsequent to the enactment of the act. The primary end in construing a statute, said the court, is to ascertain and effectuate, if possible, the legislative intent. To ascertain that intent resort must first be had to the context of the statute in question and to its subject matter, and when from the language of a statute the legislative intent is clearly apparent it must prevail. However, resort to the context and language of the statute here involved does not disclose any express statement or language from which it can be deduced whether or not the lien was intended to apply to services rendered prior to the passage of the act, for on that specific question the statute is silent and inscrutable. The court then determined the question in the light of another rule of statutory construction to the effect that retroactive legislation is looked on with disfavor and that statutes will be construed to be effective in futuro only unless the contrary intent clearly appears. The court

therefore concluded that the lien act does not operate retroactively and held that the clinic had no lien on the proceeds of the settlement for services rendered prior to the passage of the medical lien act.

In resisting the application of the rule of statutory construction against giving retroactive effect to a statute, unless the legislative intent to do so is clearly apparent, the clinic contended that the medical lien act is remedial in its nature and for that reason the rule of statutory construction just stated does not apply. It may be conceded, answered the court, that the lien act is remedial in its nature in the sense that it affords an additional remedy for the collection of a debt. However, the medical lien act is in derogation of the common law and therefore it is to be strictly construed and its operation will not be extended for the benefit of any persons who do not clearly come within its terms. Although the statute was enacted for the protection of hospitals, nurses and physicians generally it does not say, nor does it intimate, that those who perform services prior to its enactment were nevertheless entitled to its protection.

The court pointed out, however, that some of the services rendered by the clinic were performed after the statute became effective. Two roentgenograms were taken in October and November 1937, subsequent to the effective date of the lien act. These services, the court held, were wholly segregable from the services rendered at the hospital prior to the passage of the act, and the court could see no reason why the statute should not be held applicable to the latter portion of the services rendered. Accordingly, the court reversed the judgment awarding the entire $1,000 to the clinic and directed the trial court to determine the value of the roentgenograms referred to and to enter a judgment in favor of the clinic to the extent of their value.—*Layton v. Home Indemnity Co., 113 P. (2d) 538 (Wash., 1941).*

Federal Food, Drug, and Cosmetic Act: "Electreat Mechanical Heart" Misbranded.—The United States of America filed a libel in the district court, W. D. Missouri, W. D., for the destruction of six devices referred to as "Electreat Mechanical Hearts," which were seized by the government after moving in interstate commerce. The devices, the government claimed, were misbranded within the meaning of the Federal Food, Drug, and Cosmetic Act because of misleading labeling. The basis of this claim were statements made in a booklet that accompanied each seized device, that the device

> will relieve pain, strengthen weak eyes, soothe sore eyes, improve the hearing, cure earache, moisten dry noses or dry running noses, thicken thin lips, relieve toothache, strengthen the voice, relieve sore throat, build up weak lungs, relieve pleurisy, strengthen the kidneys, cure lumbago, relieve constipation, soothe the piles, is good for neuritis, subtracts pain from burns, relaxes the muscles in a stiff thumb and soothes the pain in a mashed finger, retards or accelerates the development of a boil, heals broken noses, relaxes muscle cramps, is good for varicose veins and will warm cold feet or stop the foot from perspiring.

Each booklet gave specific directions concerning the use of the device to effect the ends just stated. The device itself, as described by the court,

> consists of a cylindrical metal container having much the appearance of an ordinary flashlight, approximately ten-eleven inches long, an inch and a half in diameter with two small flashlight batteries in one end and in the other two coils. The primary coil is wound upon a soft iron core. The secondary coil is so wound that it may be moved longitudinally over the primary coil by means of a button attached to it and extending through the metal cylinder much as the switch on an ordinary flashlight is arranged. A common vibrator such as might be used on the old-fashioned doorbell, makes and breaks the current from the battery and transforms the galvanic current from the battery into the faradic or alternating current which is delivered to the body through a projection on one end of the instrument. The strength of the charge delivered to the body from the instrument is increased or decreased by means of sliding the secondary coil further over or away from the primary coil.

The manufacturer intervened in the cause and attempted to justify the numerous claims made for the device on the theory that "the instrument produces a faradic electrical current with the alternating impulses occurring at the rate of from 140 to 180 times a second which would cause the muscles and muscular tissue of the human body to contract and relax with beneficial results."

In this case, said the district court, the good faith of the manufacturer is not in issue. Nor is the inherent danger of the device or lack of it of any consequence. The issue is simply whether the claims made for the device are false or misleading. Convincing testimony, said the court, was offered by the government to demonstrate that even the principle sought to be followed by the manufacturer could not be applied with this instrument. It was pointed out that the speed or rapidity at which muscular tissue could contract and relax was much less than the rate at which the vibrations occurred in this instrument and the alternating impulses were given, and hence the only effect from the use of the instrument on the muscles of the body would be to cause them to contract and remain so until the instrument was removed, the batteries wore out or the muscles relaxed from fatigue. The extent of the accuracy of the actual claims made for the device and the literature accompanying it may be summarized much as one of the medical witnesses expressed it, when, in describing a diagram of the human anatomy with accompanying descriptive matter which appeared in one of the exhibits produced at the trial, he stated that there was an element of truth in the diagram, the element of truth being that the head was on the right end of the picture and the "rump" appeared in the proper position. From a practical standpoint, continued the court, the benefit to be derived from the use of the device was such as was stated by another witness that the use of the instrument would not injure a person if there was nothing the matter with him but that if he was suffering from any disorder or ailment its use might and probably would be injurious.

The court concluded that the claims made for the devices in the literature accompanying them were as falsely misleading as might well be possible by the use of the English language and that since the devices had moved in interstate commerce the provisions of the Federal Food, Drug, and Cosmetic Act applied. The court deemed it beyond the issues of the case to determine whether or not the manufacturer acted in good faith and honest belief that the devices would do the things claimed for them, and the court accordingly ordered the destruction of the devices. —*United States v. 6 Devices, "Electreat Mechanical Heart," 38 F. Supp. 236 (1941).*

Society Proceedings

COMING MEETINGS

American Association for the Study of Neoplastic Diseases, Washington, D. C., Sept. 4-6. Dr. Eugene R. Whitmore, 2139 Wyoming Ave. N.W., Washington, D. C., Secretary.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., Sept. 11-13. Dr. James R. Bloss, 418 Eleventh St., Huntington, W. Va., Secretary.

American Association of Railway Surgeons, Chicago, Sept. 8-10. Dr. Daniel B. Moss, 547 West Jackson Blvd., Chicago, Secretary.

American Congress of Physical Therapy, Washington, D. C., Sept. 1-5. Dr. Richard Kovacs, 2 East 88th St., New York, Secretary.

American Hospital Association, Atlantic City, N. J., Sept. 15-19. Dr. Bert W. Caldwell, 18 East Division St., Chicago, Executive Secretary.

American Roentgen Ray Society, Cincinnati, Sept. 23-26. Dr. Carleton B. Peirce, Royal Victoria Hospital, Montreal, Canada, Secretary.

Colorado State Medical Society, Estes Park, Sept. 17-20. Mr. Harvey T. Sethman, 537 Republic Bldg., Denver, Executive Secretary.

Indiana State Medical Association, Indianapolis, Sept. 23-25. Mr. Thomas A. Hendricks, 23 East Ohio St., Indianapolis, Executive Secretary.

Michigan State Medical Society, Grand Rapids, Sept. 16-18. Dr. L. Fernald Foster, 2020 Olds Tower, Lansing, Secretary.

National Medical Association, Chicago, Aug. 18-22. Dr. John T. Givens, 1108 Church St., Norfolk, Va., General Secretary.

Nevada State Medical Association, Elko, Sept. 26-27. Dr. Horace J. Brown, P. O. Box 698, Reno, Secretary.

Northern Minnesota Medical Association, St. Cloud, Aug. 15-16. Dr. Clarence Jacobson, Chisholm, Secretary.

Oregon State Medical Society, Portland, Sept. 3-6. Dr. M. L. Bridgeman, 1020 S. W. Taylor St., Portland, Secretary.

Rocky Mountain Medical Conference, Yellowstone Park, Wyo., Sept. 2-4. Mr. W. H. Tibbals, 610 McIntyre Bldg., Salt Lake City, Secretary.

Washington State Medical Association, Seattle, Aug. 24-26. Dr. Vernon W. Spickard, 1305 Fourth Ave., Seattle, Secretary.

Wisconsin, State Medical Society of, Madison, Sept. 10-12. Mr. G. B. Larson, 110 East Main St., Madison, Assistant Secretary.

Current Medical Literature

AMERICAN

The Association library lends periodicals to members of the Association and to individual subscribers in continental United States and Canada for a period of three days. Three journals may be borrowed at a time. Periodicals are available from 1931 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 18 cents if three periodicals are requested). Periodicals published by the American Medical Association are not available for lending but can be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

American J. Digestive Diseases, Huntington, Ind.

8:149-196 (May) 1941

Roentgenologic Diagnosis of Tumors of Small Bowel. H. P. Doub and H. C. Jones, Detroit.—p. 149.
Basal Secretion of Pavlov Pouch Dogs as Influenced by Oxygen Want. E. J. Van Liere and P. E. Vaughan, Morgantown, W. Va.—p. 155.
Nutrition and Defense. Agnes Fay Morgan, Berkeley, Calif.—p. 156.
Effect of Aluminum Hydroxide on Acid-Base Balance and Renal Function. J. B. Kirsner, Chicago.—p. 160.
Influence of Certain Fruit Juices on Gastric Function. H. W. Haggard and L. A. Greenberg, New Haven, Conn.—p. 163.
Secretion of Chlorine Ions in Achlorhydric Gastric Juice: Observations by Means of Radioactive Chlorine. A. Brunschwig, R. L. Schmitz and L. Slottin, Chicago.—p. 171.
Studies on Cholagogue and Choleretic Effects of Bile Acids as Compared with Oleic Acid. E. W. Lipschutz and I. A. Feder, Brooklyn.——p. 173.
Influence of Phenolphthalein on Liver. B. Fantus, F. Steigmann and J. M. Dyniewicz, Chicago.—p. 176.
Indications for Gastric Resection. F. H. Lahey, Boston.—p. 180.
Atypical Regional Ileitis: Roentgenologic Limitations. A. M. Yunich, Albany, N. Y., and B. B. Crohn, New York.—p. 185.

American J. Obstetrics and Gynecology, St. Louis

41:547-732 (April) 1941. **Partial Index**

Continuation Study: Postgraduate Education: Experiment. J. C. Litzenberg, Minneapolis.—p. 547.
Inhibition of Lactation. H. L. Stewart Jr. and J. P. Pratt, Detroit.—p. 555.
*Blood Prothrombin Levels in Newborn. C. P. Huber and J. C. Shrader, Indianapolis.—p. 566.
Pituitary Extract for Dystocia Due to Uterine Inertia in First Stage of Labor. R. S. Siddall and D. G. Harrel, Detroit.—p. 589.
Relative Effect of Analgesia and Anesthesia in Production of Asphyxia Neonatorum. H. Henderson, E. B. Foster and L. S. Eno, Detroit.—p. 596.
Conglutinatio Orificii Externi as Factor in Delayed Labor. P. J. Carter, New Orleans.—p. 606.
Study of 107 Cases of Uterine Bleeding with Endometrial Biopsies. G. F. Douglas, Birmingham, Ala.—p. 624.
*Therapeutic Value of Tubal Patency Tests in Sterility and Infertility. M. L. Leventhal and E. M. Solomon, Chicago.—p. 628.
Occurrence of Diphtheria Antitoxin in Human Pregnant Mother, Newborn Infant and Placenta. J. Liebling, G. P. Youmans and H. E. Schmitz, Chicago.—p. 641.
Cancer of Cervix Following Supravaginal Hysterectomy. G. G. Ward, New York.—p. 660.
Some Clinical Observations on Endocrinology of Abortion. E. C. Hamblen, Durham, N. C.—p. 664.
Clinical Study of Progesterone Therapy by Pellet Implantation. D. R. Mishell, Newark, N. J.—p. 687.
Incidence of Endometrial Hyperplasia with Uterine Fibroids and External and Internal Endometriosis (Adenomyosis). D. N. Henderson, Toronto, Canada.—p. 694.

Blood Prothrombin Levels in Newborn.—Huber and Shrader determined blood prothrombin levels of 506 infants. There were 15 stillborn infants and 9 infants who died during the neonatal period, giving a gross fetal mortality of 4.5 per cent. The mothers of 2 of the 9 infants received vitamin K subsequent to delivery, 4 were born of mothers who received vitamin K during labor and 3 were in the control group of 182 infants receiving no vitamin K. In 2 of these 9 infants a clinical diagnosis of cerebral hemorrhage was confirmed at necropsy; the mothers of both infants received vitamin K during labor. In the second infant a prothrombin determination following the initial evidence of cerebral irritation was 50 per cent of normal. This infant was given 2 mg. of menadione by gavage nine hours before death. Necropsy disclosed an intracranial hemorrhage as the cause of death. That a low prothrombin level in the blood of either of these infants was a factor in the intracranial hemorrhage is not justified, as in the first case there was obvious trauma and in the second the clotting activity was not impaired significantly. The production of a normal clot is only one of the factors in the control of hemorrhage. The permeability and fragility of the capillaries also play an important part. It seems fair to assume that trauma will continue to be the predominant cause of intracranial hemorrhage in the newborn. However, in an infant subjected to some degree of injury a significant hemorrhage may fail to develop unless the prothrombin level is definitely decreased, and a mild hemorrhage may become severe with the physiologic decrease in prothrombin from forty-eight to seventy-two hours subsequent to delivery. Therefore the administration of vitamin K during the early neonatal period or during labor should afford some increased protection. From a study of their data (182 infants given no vitamin K, 200 given menadione after delivery and the mothers of 117 infants given menadione during labor) the authors state that the preparation is remarkably prophylactic by either plan of administration. With some dosage adjustment and the time of its administration, complete prevention in a decrease of prothrombin may be anticipated. The data suggest that its administration more than two hours before delivery will prevent a significant decrease in more than 95 per cent of infants during the neonatal period and that all infants whose mothers have not received vitamin K more than two hours before delivery should receive 1 mg. of menadione six and twenty-four hours following delivery. All infants subjected to a prolonged or difficult delivery should be given a vitamin K preparation during the early neonatal period. The authors did not encounter a typical case of hemorrhagic disease in the newborn, but they observed 7 infants in whom various hemorrhagic tendencies were manifest. The level of clotting activity of these infants was below 10 per cent of normal. The administration of 1 mg. of menadione caused a prompt increase in the clotting activity and a cessation of hemorrhagic tendencies. More menadione may be required as determined by the prothrombin level. This group of infants offers confirmatory evidence that hemorrhagic disease is due to a prothrombin deficiency. Infants who show a prolonged low level of clotting activity without evidence of hemorrhage should be considered subclinical subjects of hemorrhagic disease. Perhaps some other factor is necessary to precipitate the hemorrhage.

Tubal Patency Tests in Sterility.—Leventhal and Solomon investigated 285 patients complaining of sterility or infertility. Conception took place in 88, or 31 per cent, of the group. Not every patient had a tubal patency test performed. When an obvious cause was discovered at the initial examination it was treated first. Thus the eradication of chronic endocervicitis with erosion or eversion was frequently followed by conception. The tubal patency test was performed on 165 patients. With male deficiencies and definite glandular dyscrasias eliminated, there remain 133 women in whom tubal insufflation or oil instillation as a therapeutic measure could be evaluated. In 114, or 85.7 per cent, patency of one or both tubes to gas or oil was demonstrated, and 51, or 45 per cent, of the latter became pregnant. Of the 19 in whom patency could not be demonstrated only 3, or 15.8 per cent, became pregnant. Altogether 54, or 40.8 per cent, of the 133 patients conceived. The average duration of marriage in the successful group was three and eight-tenths years, as compared with five and two-tenths years in the unsuccessful group. Generally accepted criteria by which the responsibility of insufflation may be estimated include pregnancy within two months without other therapeutic measures; 28 patients met these requirements. Thus, 21.5 per cent of patients on whom the tubal patency test was performed became pregnant and the pregnancy could be attributed directly to the procedure. Of these 28 patients 14 conceived immediately after the test, that is without an ensuing menstruation; 23 conceived within one month of the test. The study of the relationship of the patency test to pregnancy and associated pathologic changes among the 133 women shows that of the 63 unsuccessful cases with positive patency 27, or 42.5 per cent, showed the presence of associated pathologic changes. Thus the factor of associated pathologic conditions is an important one in influencing the therapeutic effect of insufflation and oil instillation. Only 6, or 15.8 per cent, of the successful group had associated pathologic conditions. A

total of 42 per cent of all the patients in whom the tubes were patent to gas or to gas and oil became pregnant (37 of 87). A positive illustration of the therapeutic value of insufflation alone is demonstrated by 1 patient who conceived on two occasions immediately after the Huhner and patency tests three years apart. In 6 instances with the tubes patent to gas but not to oil, pregnancy occurred only once. Thirteen of 20 patients in whom patency to gas was not demonstrated and who later evidenced patency to iodized oil became gravid, whereas none of the 9 women whose tubes were nonpatent to gas and who had no follow-up with oil conceived. This observation may be interpreted as evidence for the superiority of oil instillation over insufflation in the cure of sterility. One patient had a previous febrile abortion; examination revealed a third degree retroversion. Patency to gas failed on two occasions. Two oil instillations done four months apart failed to show "spill" after forty-eight hours. After the second instillation, pregnancy occurred without an ensuing menstruation. Similar instances occurred twice.

Am. J. Roentgenol. & Rad. Therapy, Springfield, Ill.

45:481-640 (April) 1941

*Tuberculin Test and Roentgenogram in Early Tuberculous Lesion. T. L. Badger and M. Ritvo, Boston.—p. 481.
Tuberculosis of Mediastinum. C. S. Keefer, Boston.—p. 489.
Tuberculous Tracheobronchitis: Radiation Therapy. L. F. Davenport, Boston.—p. 494.
*Sarcoid Disease as Revealed in Chest Roentgenogram. D. S. King, Boston.—p. 505.
*Incidence of Bronchomycosis in the South. R. J. Reeves, Durham, N. C.—p. 513.
Left Ventricular Aneurysm: Report of Case. W. C. Huyler, New York.—p. 521.
Calcification of Choroid Plexus and Its Displacement by Expanding Intracranial Lesions. A. E. Childe, Montreal, Canada.—p. 523.
Cancer of Stomach in the Young. G. McNeer, New York.—p. 537.
Differential Diagnosis of Intestinal Polyposis. J. Felsen, New York.—p. 551.
Roentgen Diagnosis of Cysticercus. A. I. Dombrovsky, Rostov on the Don, Soviet Union.—p. 558.
Partial Hypoplasia of Lower Jaw (Achondroplasia Partialis). K. Goldhamer, Quincy, Ill.—p. 563.
Treatment of Carcinoma of Breast by Interstitial Irradiation. R. W. Teahan, Philadelphia.—p. 567.
Adenocarcinoma, Scirrhous Type, of Left Male Breast, with Generalized Bone Metastasis: Case Report. S. K. Livingston, Hines, Ill.—p. 589.
Roentgen Treatment of Lobar Pneumonia. E. B. Settle, Rock Port, Mo.—p. 591.
Experimental Studies with "Contact" Roentgen Rays: Time-Intensity Factor of "Tumor Dose" for Rat Sarcoma 39 in Situ. J. Gershon-Cohen, H. Shay and S. S. Fels, Philadelphia.—p. 600.
Dependence of Biologic Effect of Radiation on Intensity and Wavelength, as Measured by Delayed Lethal Action on Chick Embryos. D. E. Lea, Cambridge, England.—p. 605.
Production and Measurement of Soft Roentgen Rays for Biologic Experiments. D. E. Lea, Cambridge, England.—p. 614.

Tuberculin Test and Roentgenogram in Early Tuberculosis.—Badger and Ritvo state that a study of tuberculosis in nurses at the Boston City Hospital, carried on since 1932, showed that the morbidity is almost equally distributed between those who enter training tuberculin positive and those who enter tuberculin negative. All but a few of the nurses who enter training with a negative tuberculin test become positive during their three years of training. In the majority of these the usual benign primary infection develops in a region not demonstrable roentgenographically. In a few nurses small but definite roentgenographic lesions develop without clinical manifestations, while others progress to clinically active tuberculosis. Once an early tuberculous lesion is established by its persistence in the roentgenogram it must, if possible, be determined whether it is an old healed scar, a latent process potentially active in the future, an early destructive infiltration or a benign lesion which will heal spontaneously. It is the roentgenologist's responsibility to recognize these small tuberculous infiltrations. He must understand their latent potentialities and in cooperation with the internist interpret correctly their characteristics.

Sarcoid Disease as Revealed in Roentgenogram.—From the 142 cases of established sarcoid disease encountered in three hospitals since 1921, King selected for discussion 50 presenting characteristic shadows in chest roentgenograms. In 24 diagnosis was proved by biopsy of the skin or a node, in 11 there were sarcoid lesions of the eyes, skin, glands or bones, but no biopsy, and in 15 only the chest roentgenogram constituted the evidence. Roentgenograms are considered characteristic when the process is confined entirely or mainly to hilar nodes, when it involves only the pulmonary fields giving an appearance indistinguishable from miliary tuberculosis, or when it involves the nodes and apparently radiates from them into the pulmonary fields. The roentgenogram of the chest is not a measure of the gravity of the disease; extensive pulmonary changes may clear without any respiratory symptoms and be accompanied by only slight evidence of a disseminated process; or there may be little involvement of the chest roentgenologically, but lesions in other organs may cause prolonged and serious disability. The disease is of a protean nature. Pulmonary lesions are only part of the whole picture of sarcoidosis; nevertheless they are important and one must try to discover what happens to them with the passage of time. A satisfactory follow-up was made of 37 cases. In 23 the lesions seen in the chest have cleared completely, or almost completely, in from seven weeks to three years, the average time being twenty-two months. In 3 the process is clearing but has not disappeared entirely after from one to three years. In 8 there is no change after from three months to four years. In 3 the process is spreading after seven months, eight months and four years, respectively. In the last of these after apparent clearing there was a relapse with marked roentgen involvement of the lungs but without a corresponding increase in symptoms. This is the author's only instance of relapse after apparent clearing. The process may again clear, but it is possible that it is now active pulmonary tuberculosis. He has observed only 2 cases of possible pulmonary fibrosis suggesting that sarcoid may be the underlying etiologic factor. Generally sarcoid lesions of the chest tend to clear, but the time required varies and may be long. These lesions must be differentiated from erythema nodosum and mycotic infection.

Bronchomycosis in the South.—Reeves reports 79 cases of bronchomycosis, of which 16 were due to blastomycosis, 10 to actinomycosis, 40 to moniliasis, 3 to sporotrichosis, 5 to aspergillosis, 2 to geotrichosis, 2 to coccidioidal granuloma and 1 to torulosis. There were 47 males and 32 females. The age range was from 4 months to 66 years and the duration of symptoms from one month to fifteen years. Twenty-six of the patients are living. The roentgen appearance in the early stages resembles bronchitis or pneumonitis rather than tuberculosis, but as the fungi give rise to the formation of pus within the alveoli either a bronchiectasis or consolidating masses result within the pulmonary structure. The Monilia infection seemed to present the most consistent roentgen changes; in the early stages the lesions were diffuse with small nodules throughout the entire pulmonary field, and in the later stages the lungs presented a diffuse chronic interstitial fibrosis extending from the hilus throughout the lungs, with less involvement of the apexes. This can usually be differentiated from tuberculosis because the fibrosis is more of a network with heavy beading. About 60 per cent of the actinomycosis cases showed pulmonary and skeletal involvement. The pulmonary lesions are usually pneumonic with irregular areas of consolidation which often break down and suppurate, leaving large cavities. It often starts as a central pneumonic process and occasionally extends through the chest wall. The author's patient with such lesions has been well for nine years. The pulmonary lesions of blastomycosis vary from dense pneumonic masses to a miliary distribution. The roentgen appearance of aspergillosis was consolidation simulating pneumonia. Geotrichosis has no typical roentgen appearance. The lungs, when involved, resemble chronic tuberculosis with heavy studding along the bronchi; frequently cavitation occurs. Sporotrichosis seldom produces pulmonary lesions. In 1 patient there was a diffuse nodular infiltration, radiating out along the bronchi in both lungs. The lesions have been reported as being more often a dense gummatous mass with long mycelia invading the pulmonary structure. Torulosis has no typical roentgen appearance. The lesions are often compatible with tuberculosis except that cavitation is rare. Bronchomycosis should be considered one of the more common problems of differential diagnosis. The presence of fungi only in the sputum is not sufficient to establish a diagnosis. Many fungi in the sputum are not virulent or pathogenic. Fungi may be pathogenic but still not the cause of the disease in question.

As yet many institutions are not adequately equipped for the isolation of the fungi, entailing time and expense; only the presence or absence of tubercle bacilli is looked for. Bronchomycosis is not universally fatal; cure is sometimes possible.

American Review of Tuberculosis, New York

43:449-580 (April) 1941

Tuberculosis in the Rat: I. Gross Organ Changes and Tuberculin Sensitivity in Rats Infected with Tubercle Bacilli. C. C. Wessels, Philadelphia.—p. 449.

Id.: II. Fate of Tubercle Bacilli in Various Organs of Rat. C. C. Wessels, Philadelphia.—p. 459.

Artificial Pneumothorax in Patients Over 40. S. Diamond and H. T. Ivey, Legion, Texas.—p. 475.

Tuberculosis Case Finding: X-Ray Survey of 28,331 Trade Union Members in New York City, 1939. H. R. Edwards, Sally Preas, Jean Downes and A. Robins, New York.—p. 491.

Diameters of Normal and Abnormal Bronchi: Statistical Study. J. Chapman, Sanatorium, Texas, and H. E. Smith, Austin, Texas.—p. 504.

*Bronchiectasis: Morphologic Varieties of Bronchiectasis in Relation to History, Symptoms and Signs. J. Chapman, Sanatorium, Texas, and H. Hoskins, San Antonio, Texas.—p. 512.

Classification of Chronic Bronchiectasis. J. Chapman and D. McCullough, Sanatorium, Texas.—p. 521.

Comparison of Reactions to Different Tuberculins in Same Individuals. C. L. Savage, Richmond, Va.—p. 527.

Tuberculin Tests with Old Tuberculin and Purified Protein Derivative: Comparative Study of Old Tuberculin and Purified Protein Derivative in Tuberculin Testing of Guinea Pigs in Routine Diagnostic Service. E. M. Medlar, K. T. Sasano, D. W. Caldwell and E. L. Needham, Mount McGregor, N. Y.—p. 534.

*Gastric Lavage in Adults with Pulmonary Tuberculosis. W. H. Roper, Sanatorium, N. C., and W. H. Ordway, Mount McGregor, N. Y. p. 543.

Psychologic Factors in Tuberculous Patients: Observations at Sunnyside Sanatorium, with Especial Reference to Personality. I. T. Shultz, Indianapolis.—p. 557.

Bronchiectasis.—Chapman and Hoskins analyze 116 cases of bronchiectasis with regard to relationship between etiology, symptoms and signs and the type of dilatation observed on roentgen study. There were 60 instances of cylindric, 26 of saccular, 24 of varicose, 4 of fusiform and 2 of bronchiolectatic dilatation. Study of the age and sex of the patients having the principal three types fails to show any marked differences. The ratio of women to men is 3 to 2, but this approximates the proportion of the two sexes as they are admitted to the sanatorium. Blood spitting is more common with saccular dilatation. Of all the patients, equally divided among the three types, 42 per cent brought up sputum for more than two years. About one half estimated the amount at 1 ounce (30 cc.) or less in twenty-four hours. About 40 per cent denied having had pneumonia. The incidence of pneumonia in saccular bronchiectasis was greater than in the cylindric type. Influenza was denied by 27 per cent. There was no significant difference in its incidence among the three types. Nasal disease was a complaint of 35 per cent. Its incidence was about the same in the three types. Physical signs in cylindric bronchiectasis were absent more often than in the other types. The only patients known to have asthma were in the cylindric group. The authors' figures for distribution of the disease differ from other reports in that there was a relatively higher incidence in the right lower lobe as compared with the left. This is unexpected in view of the fact that congenital bronchiectasis should involve the left lower lobe more commonly. It appears that distinction between cylindric and saccular bronchiectasis is based on sound statistical grounds. This is not the case with other morphologic types.

Gastric Lavage in Adults with Pulmonary Tuberculosis.—Roper and Ordway studied fasting gastric contents of 135 tuberculous patients. Bacilli were recovered from the sputum of only 34.8 per cent of the patients. The addition of gastric lavage almost doubled the proportion, the percentage being 63. This increase was due largely to positive guinea pig inoculations with gastric contents from patients with minimal and moderately advanced disease who were unable to expectorate, and to a slightly less degree to positive lavages from patients whose sputums were negative. Excluding 12 patients with no sputum or with negative sputum who were not submitted to gastric lavage because they refused it or left the sanatorium prematurely, positive recovery was actually increased to 69.1 per cent in the 123 cases in which all tests were given. Of 78 tuberculous patients tested by the gastric lavage during the first month of residence at the sanatorium 43 had no expectoration, and smear examinations of several sputums from each of the remaining 35 had been repeatedly negative. Eleven of the 78 patients were subsequently found to be positive by guinea pig tests of the sputum, while 44 were positive by the gastric lavage alone. Thirty-six of these 44 were either not expectorating or were producing sputum which was consistently negative by microscopic examination and animal inoculation. Had lavage of the stomach been omitted, the diagnosis could not have been confirmed in 36, or almost one half, of the 78 patients. The test is of assistance not only in the diagnosis but also in the management of the tuberculous patient. After years of treatment the sputum may disappear or become negative, while the gastric contents still exhibit virulent tubercle bacilli. Diagnosis of tuberculosis on the basis of smear examinations is not dependable. A single negative gastric lavage does not exclude active tuberculosis, for the same variability occurs as with sputum; the tuberculous patient may contain many tubercle bacilli on one day and none on the following day, or many tubercle bacilli may be present in a morning specimen with none being raised in the afternoon of the same day.

Annals of Internal Medicine, Lancaster, Pa.

14:1741-1968 (April) 1941

Some Aspects of Nature of Chemical Changes Occurring in Atheromatosis. I. H. Page, Indianapolis.—p. 1741.

Cholesterol Content of Renal Arteries and Thoracic Aorta in Relation to Hypertension and Vascular Pathology. M. Bruger and M. R. Chassin, New York.—p. 1756.

Nature of Malignant Hypertension. H. A. Derow and M. D. Altschule, Boston.—p. 1768.

*Hepatolenticular Degeneration (Wilson's Disease) Following Splenectomy; Interrelationship of Reticuloendothelial and Central Nervous System. A. M. Rabiner, H. Joachim and I. S. Freiman, Brooklyn.—p. 1781.

Quantitative Serologic Test for Syphilis; Its Variability, Usefulness in Routine Diagnosis and Possible Significance: Study of 1,665 Cases. J. E. Moore and H. Eagle, Baltimore.—p. 1802.

Causes of Secondary Fever in Sulfapyridine Treated Pneumonia. H. F. Dowling and T. J. Abernethy, Washington, D. C.—p. 1815.

*Treatment of Pneumococcic Pneumonia with Sulfapyridine. W. L. Winters, P. S. Rhoads, W. W. Fox and R. Rosi, Chicago.—p. 1827.

Acquired Hemolytic Icterus. W. M. Fowler, Iowa City.—p. 1838.

Intravenous Streptococcus Vaccine Treatment of Chronic Rheumatoid Disease. M. Wetherby, Minneapolis.—p. 1849.

*Metabolic Craniopathy: Hyperostosis Frontalis Interna. P. T. Knies and H. E. Le Fever, Columbus, Ohio.—p. 1858.

Role of Diabetes in Development of Degenerative Vascular Disease, with Special Reference to Incidence of Retinitis and Peripheral Neuritis. T. J. Dry and E. A. Hines Jr., Rochester, Minn.—p. 1893.

Hepatolenticular Degeneration Following Splenectomy.—Rabiner and his associates report 2 instances of a heretofore unrecorded sequel to splenectomy. Symptoms indicating hepatolenticular degeneration followed soon after a splenectomy performed for anemia and purpura associated with splenomegaly. The symptoms direct attention to a possible contraindication to a splenectomy. In the first case mild parkinsonian tremors of four years' duration followed a febrile illness diagnosed as typhoid. The tremors were most likely due to striatal disease dating back to the febrile illness of four years before. This tremor was not associated with any gross disability. Following the splenectomy the tremor and the muscular rigidity were soon extreme, and the Kayser-Fleischer ring was noticed for the first time. The neurologic disorder then progressed rapidly. The splenectomy probably aggravated the striatal disease. The other patient had a chronic hemorrhagic diathesis for many years. Headaches, blurred vision and slowing up of activities suggested that the symptoms were due to epidemic encephalitis. However, none of the objective symptoms of disease of the basal ganglions were present until two months after the splenectomy, when the entire neurologic syndrome was initiated and progressed rapidly and resembled hepatolenticular disease. For a time the course remained stationary. Almost two years after the operation, in a fit of despondency, the first patient committed suicide. Necropsy revealed typical features of Wilson's disease. While the changes were diffuse, they were far more marked and advanced in the basal ganglions, thalamus and hypothalamus. A large number of type II glial cells of Alzheimer, typical of Wilson's disease and pseudosclerosis, were found in these areas. The authors feel that the case must be

placed in the category of hepatolenticular degeneration. While it does not fulfil the original pathologic criteria of Wilson, cystic degeneration of the putamen, nevertheless degeneration in this region, with formation of a spongy state, was prominent and the friability led them to believe that cystic degeneration might ultimately have ensued. The changes, while diffuse, did not follow the typical description of pseudosclerosis, which include diffuse and widely scattered Alzheimer cells. The authors supposed that they were dealing with a form of hepatolenticular degeneration intermediate between Wilson's disease and pseudosclerosis. It is apparent that removal of the spleen was not the primary etiologic factor responsible for the onset of hepatolenticular disease, as in each case the disease had existed preoperatively. This was shown by cirrhosis of the liver found at operation and by the preoperative existence of minimal neurologic symptoms the significance of which had been overlooked. It was present in a latent or subclinical form, and operation accelerated its process.

Sulfapyridine for Pneumococcic Pneumonia.—Winters and his colleagues from Jan. 15 to May 15, 1939 treated 123 pneumococcic pneumonia patients with sulfapyridine, and the mortality was 11.4 per cent. The mortality among 25 patients who received neither sulfapyridine nor antipneumococcus serum was 25 per cent. Study of the 123 treated patients shows that sulfapyridine was effective against all types of pneumococci encountered (eighteen types) and that its use was relatively safe. No deaths could be attributed to the treatment. Nausea and vomiting occurred frequently (35 per cent) but was serious in only 4 per cent and necessitated discontinuance of treatment. A study of the fatalities indicates that the most important prognostic factor was delay in treatment. Age, extent of pulmonary involvement, bacteremia and alcoholism were, in order of importance, further determining factors. Serum was not administered until it appeared obvious that sulfapyridine alone was inadequate, as indicated by continued fever, prostration, persistent bacteremia and spread of pneumonia. Toward the end of the study it was felt that serum should be given at once when the duration of the pneumonia was of four or more days, when more than one lobe was involved, when alcoholism was evident and when beginning septic complications, diabetes, heart disease or other diseases were present. When serum was used the initial dose was 200,000 or more units, and larger doses were given when more than one of the foregoing factors were present. Further serum treatment depended on the appearance of the patient and on the presence or absence of agglutinins in the patient's blood. The authors now feel that, whether any of the foregoing factors are present or not, patients whose admission blood culture is positive should be given serum unless the improvement to sulfapyridine between the drawing of the blood for the culture and the positive culture has been striking. While a few fatal cases are to be expected, the goal should be to lower this "irreducible minimum" to less than 5 per cent. This probably can be achieved through the persistent application of the foregoing principles of treatment.

Metabolic Craniopathy.—Knies and Le Fever review the literature on metabolic craniopathy for the past one hundred and seventy-three years, extend the concept of the malady and suggest additional factors. The disease has been linked with the pituitary gland and occasionally with ovarian menopausal changes because of the average age of the affected patients. However, 5 of the authors' patients were less than 20 years of age and 8 less than 30, making it apparent that the age of pathogenesis may long precede the menopause. Two cases have been reported under the age of 20 years. A similar doubt as to the menopausal effect is cast by the considerable number of cases of males reported in the literature. There were 5 males among the authors' series of 28. Three of their juvenile patients (2 male and 1 female) were siblings of a mother with well developed frontal hyperostosis, suggesting a familial and possibly a hereditary basis. Their cases are the first to give evidence of such a background. Sex preponderance is suggested by these instances. The usual discovery of the disease at later ages may depend on slowly progressive changes, beginning earlier with secondary changes which are detected later. The discovery of well marked hyperostosis in traumatic cases without previous symptoms suggests a possible long latent period. The slight impairment of health in the earlier stages indicates that the majority of pathologic studies have been made late in senile patients and that the younger patients have been overlooked. The endocrine aspects of the disease appear unquestionable and are compatible with a hereditary concept of pathogenesis. The progress of the cranial changes of 2 patients, including not only the diploic changes but also those of frontal internal hyperostosis, has been observed by the authors for more than two years. Extracranial changes are rare. The association of trauma appears definite. It is probable that in some instances pituitary or adjacent changes induced by injury to the head have through secondary endocrine mechanisms ultimately caused hyperostosis and other features of metabolic craniopathy. In many other cases injury only brought to light previously latent, asymptomatic changes. The social implications of the disease are important; 3 instances of violent antisocial tendencies have been reported, including a patient with "revenge psychosis" and 2 murderers reported by the authors. Usually the psychoses are mild and tend to be intermittent. Thus the disease assumes further importance in considering the background of psychoses of uncertain etiology, as demonstrated by Moore. Treatment has been symptomatic or empiric and generally ineffective. In view of the pathogenesis, surgical removal of the hyperostoses, difficult because of the dense dural adhesions, would probably not be either feasible or effective. Furthermore the clinical features are not always dependent on the type or extent of the osseous changes and are often dissociated in terms of time. Future reports should include critical analyses of the treatment and detailed study of the familial background.

Connecticut State Medical Journal, Hartford

5:317-402 (May) 1941

Differential Diagnosis of Low Back Pain. P. P. Swett, Bloomfield.—p. 330.
Connecticut Doctors in Other Fields. H. Thoms, New Haven.—p. 337.
Sulfathiazole Treatment of Gonococcic Infections in Ambulatory Patients. L. E. Celentano and R. M. Lewis, New Haven.—p. 344.

Delaware State Medical Journal, Wilmington

13:51-68 (April) 1941

Atheromatous Cardiovascular Disease: Newer Concepts in Diagnosis and Treatment. J. B. Wolffe, Philadelphia.—p. 51.
Behavior Problem in Children. A. Gordon, Philadelphia.—p. 58.

Indiana State Medical Assn. Journal, Indianapolis

34:175-234 (April) 1941

Industrial Hygiene Activities of Indiana State Board of Health. L. W. Spolyar, Indianapolis.—p. 175.
Medical Relationships in Industry. C. M. Peterson, Chicago.—p. 180.
Medical Practice in Industry. E. S. Jones, Hammond.—p. 182.
Occupational Dermatitis. F. M. Gastineau, Indianapolis.—p. 184.
Industrial Health and Safety Precautions. J. L. Ridinger, East Chicago. p. 188.
Health and Income. A. J. Fritz, Indianapolis.—p. 191.
Problems of Medicolegal Relationships. A. F. Knoefel, Terre Haute. —p. 193.
Physiologic Pathology of Shock as Applied to Treatment. A. S. Giordano, South Bend.—p. 197.
Cardiovascular Equilibrium During Anesthesia. E. A. Rovenstine and S. J. Martin, New York.—p. 201.
Present Day Aspects of Etiology of Cancer. N. K. Forster, Hammond. —p. 208.
Early Symptoms and Signs of Cancer of Uterus. J. F. Habermel, New Albany.—p. 211.

34:235-286 (May) 1941

Activities of the Committee on Conservation of Vision. E. L. Van Buskirk, Lafayette.—p. 235.
Two Important Developments in Ophthalmology. R. J. Masters, Indianapolis.—p. 237.
The Eye in Industry. E. O. Alvis, Indianapolis.—p. 239.
Trachoma. N. S. McBride, Terre Haute.—p. 245.
Glaucoma: Associated Headaches. C. P. Clark, Indianapolis.—p. 247.
Significance of Blood Eosinophilia in Group of Ambulatory Patients. B. Kraft, Indianapolis.—p. 249.
Doctors Watch the Ramparts, Too (Few Notes for Those Who Will Serve). J. R. Phillips, Michigan City.—p. 251.
Accidental Death. M. C. Topping, Terre Haute.—p. 257.

Journal of Bone and Joint Surgery, Boston

23:225-496 (April) 1941. **Partial Index**

Fractures in Neck of Femur in Children, with Particular Reference to Aseptic Necrosis. B. Carrell and W. B. Carrell, Dallas, Texas.—p. 225.
*Treatment of Tendons in Compound Injuries of Hand. S. Bunnell, San Francisco.—p. 240.
Subfascial Hematoma as Complication of Crushing Injuries to Foot. H. M. Childress, Jamestown, N. Y.—p. 251.
Slipping of Upper Femoral Epiphysis: End Results After Conservative Treatment. M. Forrester-Brown, Bath, England.—p. 256.
Epiphysial Dislocations in Scurvy. W. Scott, Hollywood, Calif.—p. 314.
Study of Paralytic Scoliosis Based on 500 Cases of Poliomyelitis. P. C. Colonna and F. vom Saal, Oklahoma City.—p. 335.
Osteochondromatosis. C. L. Wilmoth, Pittsburgh.—p. 367.
Anatomic Variations and Roentgenographic Appearance of Low Back in Relation to Sciatic Pain. T. A. Willis, Cleveland.—p. 410.
Neuroanatomic and Physiologic Aspects and Significance of Sciatica. W. M. Craig and M. N. Walsh, Rochester, Minn.—p. 417.
*Conservative Treatment of Sciatic Pain in Low Back Disability. J. G. Kuhns, Boston.—p. 435.
*Posterior Protrusion of Lumbar Intervertebral Disks. J. S. Barr and W. J. Mixter, Boston.—p. 444.
*Low Backache and Sciatic Pain Associated with Spondylolisthesis and Protruded Intervertebral Disk: Incidence, Significance and Treatment. H. W. Meyerding, Rochester, Minn.—p. 461.
Relief of Low Back Pain and Sciatica by Release of Fascia and Muscle. C. H. Heyman, Cleveland.—p. 474.
Fascial Elements in Associated Low Back and Sciatic Pain. A. H. Freiberg, Cincinnati.—p. 478.
Articular Facets in Relation to Low Back Pain and Sciatic Radiation. C. E. Badgley, Ann Arbor, Mich.—p. 481.

Treatment of Tendons in Compound Injuries of Hand.—According to Bunnell, the outcome of compound injuries of the hand and the prevention of sloughing tendons, infected joints and osteomyelitis is determined by (1) early operation, (2) thorough débridement and (3) covering of all vulnerable tissues. It is preferable to suture tendons within six hours after the accident, but when all factors (infection, degree of trauma, condition of patient and management) are favorable primary suture of tendons can be successfully done after six hours, in a gradually decreasing number of cases, up to but never after twenty-four hours. Tendons should never be sutured if the wound is already infected, if more than twenty-four hours old or in the presence of too much crushed, damaged, dirt-ground or badly contaminated tissue. The operation should never be done with poor hospital facilities or by an unskilled surgeon. It is preferable to leave a severed tendon undisturbed, repairing it later under good conditions, than to repair it under adverse conditions and make the limb worse. When making incisions, one should take care to avoid crossing flexion creases at a right angle or injuring small nerves. Catgut is the least satisfactory suture material. Fine stainless steel wire number 34 or 35 provokes the least reaction of all suture materials. In the primary repair of tendons, the stitch should be the simplest possible. It must cross some fibers for a spliced effect, but this should be minimal. A core suture is less likely to cause adhesions than one on the surface of the tendon. The smooth glistening epitenon surrounding the tendon should not be even scratched. The suture material should be sunk into the tendon. There should be no slack. Some central necrosis will result from the tightness, but eventually it will be replaced by tendon cells. To prevent tendon ends from pulling apart more reliance should be placed on splinting, with joints flexed to relax the tendons, than on the strength of the suture. In three weeks physiologic union allows removal of the splint, a half circumference plaster of paris. At this time, after tendon union, the suture is withdrawn and irritation from it is obviated.

Treatment for Sciatic Pain Due to Low Back Disability.—Kuhns discusses the conservative treatment and the results in 1,000 cases of sciatic pain and low back disability encountered during the last ten years. Conservative treatment includes rest in special positions, support, medications and physical therapy but not manipulation or operative procedures. The low back pain of 519 patients had been present for two weeks or less and of 481 it was of longer duration. In most instances the lesion causing the low back pain is presumed to be a ligamentous tear or a separation of fibers at the musculotendinous junction. Pain and disability often disappear after immobilization and support with attempted relaxation of the injured part. The best method of securing immobilization and ligamentous relaxation is to keep the patient constantly on a firm bed with the entire body horizontal except for slight flexion at the hip joint. This can be secured in the supine position by placing a pillow under the knees or, in the prone position, under the abdomen. If the ligamentous injury is severe it may be necessary to apply a plaster back shell or a plaster spica for immobilization. In most instances while the patient is in bed physical therapy, heat daily, should be given to the posterior surface of the spine and pelvis to improve local circulation, relax muscular spasm, relieve pain and hasten the absorption of inflammatory exudate. As soon as muscular spasm and pain begin to subside and the patient can move in the bed without much discomfort, exercises to teach him muscular coordination are begun. The average period of complete recumbence was ten days. It should be continued until pain and local tenderness subside. This is not the end of treatment, as usually a prolonged convalescence is required for the individual to be able to resume his usual activities. Because the time required to heal a ligamentous tear is long and an unguarded movement during convalescence may cause it to recur, a spinal support is advisable. For the author's patients an average of forty-eight days was required before the spinal symptoms and signs subsided and the patients returned to work. The extremes were one week and ten months. Some patients who return and continue at work are never wholly free of symptoms, although physical signs do not suggest a spinal lesion. The reasons for their continued symptoms may be a chronic arthritis, a congenital anomaly making the back potentially weak, working long hours with the back bent or the inability to learn to use the body in a more correct habitual position. Of the 1,000 patients treated conservatively for the relief of local and referred pain, the pain of 771 was relieved, that of 72 was not relieved and the result in 157 is not known. Of 449 patients with sciatic radiation of pain, 356, or 79 per cent, were relieved. The study suggests that all patients with acute traumatic and chronic postural strains of the low back can be expected to recover under conservative treatment. The length of treatment will depend on the severity of the trauma, the chronicity of the lesion and the cooperation of the patient. For those patients with structural changes in the vertebrae or their contiguous structures when structural restoration cannot be expected, as in metastatic malignant growths, conservative therapy may prolong function and is a definite palliative measure. If normal osseous contour is established, conservative therapy is necessary for the recovery of normal function. For the group of patients with low back pain from pressure or irritation within or about the spinal canal, conservative treatment may be of little benefit. If a definite lesion exists, its removal is indicated. The author had no patients with pain referred to the back from other parts of the body, usually the abdominal and pelvic viscera, but says that the lesion that causes the referred pain should be treated.

Posterior Protrusion of Intervertebral Disks.—Barr and Mixter find posterior protrusion of one of the lumbar intervertebral disks into the spinal canal to be the most common mechanical derangement of the low back in patients suffering from intractable sciatic pain. In addition, there may be associated thickening of the ligamentum flavum, chronic adhesive arachnoiditis, hypermobility of the involved vertebrae and edema of the involved nerve roots. Although the etiology of posterior disk protrusions is not clear, trauma to and/or degenerative changes in the intervertebral disks seem to be the usual causes. Conservative treatment should be tried in every suspected protrusion of the intervertebral disks unless there is obvious serious nerve root pressure shown by sensory or motor disturbance. Bed rest and immobilization of the lumbar spine in a plaster jacket are effective means. Surgical treatment removes the ruptured disk fragment through as small a laminectomy incision as possible. Spinal fusion at the time of the laminectomy seems to give definitely better results than laminectomy alone. Of 94 cases of proved ruptured intervertebral disk that the authors followed for at least one year after operation, 77 per cent were completely relieved of sciatic pain and in an additional 18 per cent only minor leg pain persisted. There were 2 proved cases of recurrent ruptures. The relief of back symptoms was not as satisfactory as the relief of radiating leg pain. Of the

patients in whom the spine was fused, 73 per cent and 52 per cent of those without fusion had no back symptoms. The rest had complaints of backache or weakness of varying severity. The end result of 20 proved cases, in which compensation was involved and the patients' medical expenses were paid by an insurance company, reveals that 9, or 45 per cent, have resumed their original occupation and consider themselves fully recovered, 6 others are at work at lighter jobs and 5 are still receiving compensation or have litigation pending and consider themselves disabled.

Low Backache and Sciatic Pain.—To determine the incidence of low back pain and sciatica among patients suffering from spondylolisthesis, Meyerding reviewed the histories of the 745 patients for whom such a diagnosis was made between 1918 and 1940. He found 80 patients, or 10.7 per cent, who were so afflicted. A much larger percentage of patients suffering from spondylolisthesis had vague referred pain and paresthesia of the buttocks, hips and thighs that were aggravated by activity. He believes that patients with spondylolisthesis are more likely to have a protruded intervertebral disk than are those with more stable spinal columns. Trauma was a definite factor in 43 of the 80 patients. In the author's opinion fusion of the lumbosacral region is desirable in those cases of spondylolisthesis with symptoms of protruded disk in which the surgeon is unable to demonstrate the disk at the time of the operation. This fixation will prevent movement and slipping and additional symptoms of backache and sciatic pain. Cooperation of the roentgenologist, neurologist and orthopedic surgeon has made it possible to diagnose the condition accurately and to relieve patients whose ailment has heretofore baffled the diagnostic efforts of even the most skilled surgeons.

Journal of Experimental Medicine, New York

73:571-680 (May) 1941

Blood Plasma Protein Production as Influenced by Various Degrees of Hypoproteinemia and by Amino Acids. S. C. Madden, A. P. Turner, A. P. Rowe and G. H. Whipple, Rochester, N. Y.—p. 571.

Studies on Influenza Virus: Complement Fixing Antigen of Influenza A and Swine Influenza Viruses. E. H. Lennette and F. L. Horsfall Jr., New York.—p. 581.

Quantity of Irradiated Nonvirulent Rabies Virus Required to Immunize Mice and Dogs. L. T. Webster and J. Casals, New York.—p. 601.

Organ Specificity of Tissues of Dog and Man as Shown by Passive Anaphylaxis in Guinea Pigs. G. H. Bailey and S. Raffel, Baltimore.—p. 617.

Production of Bactericidal Substances by Aerobic Sporulating Bacilli. R. J. Dubos and R. D. Hotchkiss, New York.—p. 629.

Virus from Cases of Atypical Pneumonia: Relation to Viruses of Meningopneumonitis and Psittacosis. M. D. Eaton, M. Dorthy Beck and H. E. Pearson, Berkeley, Calif.—p. 641.

Isolation of O Specific Substance from Gastric Juice of Secretors and Carbohydrate-like Substances from Gastric Juice of Nonsecretors. E. Witebsky and N. C. KlenDshoj, Buffalo.—p. 655.

Fatal Infection of Irradiated White Mice with European Typhus by Intra-Abdominal Route. P. Y. Liu, J. C. Snyder and J. F. Enders, Boston.—p. 669.

Journal of Infectious Diseases, Chicago

68:97-192 (March-April) 1941

Studies on Anaphylaxis in Mouse. R. S. Weiser, O. J. Golub and Dorothy M. Hamre, Seattle.—p. 97.

Cultures of Dysentery Group Isolated in Western Michigan. Mary Branston and Grace Eldering, Grand Rapids, Mich.—p. 113.

Primary Isolation of Small Colony Strains of Eberthella Typhosa from Blood, Feces, Urine and Sputum. Janie F. Morris, T. F. Sellers and A. W. Brown, Atlanta, Ga.—p. 117.

New Salmonella Type: Salmonella Havana. F. Schiff and I. Saphra, New York.—p. 125.

Distribution of Hemolytic Streptococci Groups A, B and C in Human Infections. L. A. Rantz and C. S. Keefer, Boston.—p. 128.

Quantitative Studies on Neutralization of Purified Papilloma Virus: I. Relations Between Serum, Total Virus and Free Virus. W. R. Bryan and J. W. Beard, Durham, N. C.—p. 133.

Failure of Treatment of Experimental Poliomyelitis with Potassium Chlorate. S. O. Levinson and A. Milzer, Chicago.—p. 171.

Growth Factors for Haemophilus Influenzae and Haemophilus Parainfluenzae. A. Bass, S. Berkman, F. Saunders and S. A. Koser, Chicago.—p. 175.

Comparisons of Clon of Plasmodium Cathemerium with Its Parent Strain and with Two Strains Derived from the Clon by Mosquito Transmission. C. G. Huff, Chicago.—p. 184.

Three Strains of Poliomyelitis Virus Isolated from Feces During the 1939 Buffalo and Detroit Epidemics. J. E. Kempf and M. H. Soule, Ann Arbor, Mich.—p. 188.

Journal of Pharmacology & Exper. Therap., Baltimore

71:301-410 (April) 1941. **Partial Index**

Cooling Drugs and Cooling Centers. F. E. Rosenthal, Leicester, England.—p. 305.

Cyverine Hydrochloride, New Synthetic Papaverine-like Compound: Report on Its Vasodilator Action in Chronic Occlusive Peripheral Vascular Diseases. R. J. Popkin, Los Angeles.—p. 320.

Fibrillation Threshold After Administration of Digitalis and Ouabain. R. Wégria, J. H. Geyer and B. S. Brown, Cleveland.—p. 336.

Reduction of Neoprontosil by Tissues in Vitro. F. Bernheim, Durham, N. C.—p. 344.

Studies in Absorption, Distribution and Elimination of Alcohol: VIII. Diuresis from Alcohol and Its Influence on Elimination of Alcohol in Urine. H. W. Haggard, L. A. Greenberg and R. P. Carroll, New Haven, Conn.—p. 349.

Id.: IX. Concentration of Alcohol in Blood Causing Primary Cardiac Failure. H. W. Haggard, L. A. Greenberg, L. H. Cohen and N. Rakieten, New Haven, Conn.—p. 358.

Action of Vitamin C and Phenol Oxidase in Inactivation of Beta Phenylisopropylamine (Amphetamine). K. H. Beyer, Madison, Wis.—p. 394.

Journal of Urology, Baltimore

45:647-794 (May) 1941. **Partial Index**

Significance of Sensory Paralyses in Poliomyelitis Patients in Whom Renal Calculi Develop. M. L. Boyd, Atlanta, Ga.—p. 647.

*Response of Cardiovascular System to Intravenous Fluids. F. D. Murphy, Milwaukee.—p. 654.

Medical English: Short Criticism of Composition and Delivery of Manuscripts. E. Rupel, Indianapolis.—p. 664.

Nephrectomy for Renal Tuberculosis After Four Stages of Thoracoplasty. G. Sewell, Detroit.—p. 669.

Malignant Tumors of Testicle. J. K. Ormond and C. L. Prince, Detroit.—p. 685.

Plastic Procedure for Correction of Hypospadias. R. M. Nesbit, Ann Arbor, Mich.—p. 699.

Diverticulum of Female Urethra. W. J. Engel, Cleveland.—p. 703.

Resection of "Large" Prostate: Technic and Results. C. D. Creevy, Minneapolis.—p. 715.

Urinary Calcium Precipitability in Medical Management of Calcium Urolithiasis. R. H. Flocks, Iowa City.—p. 721.

Use of Sulfapyridine and Sulfathiazole in Gonorrhea: Preliminary Report. D. K. Hibbs, A. A. Day, Ruth W. Jung and J. M. Brady, Chicago.—p. 727.

Importance of Identifying Urea Splitting Bacteria: Report of Clinical Experience. T. L. Schulte and G. J. Thompson, Rochester, Minn.—p. 733.

Bacterial Urease: I. Critique of Methods Heretofore Used for Demonstrating Bacterial Urease and Presentation of Valid and More Sensitive Test. E. C. White and Justina H. Hill, Baltimore.—p. 744.

Id.: II. Study of Ureolytic Action of Bacteria of Significance in Genitourinary Infection. Justina H. Hill and E. C. White, Baltimore.—p. 749.

Vasoligation and Vasotransection Without Scrotal Incision. R. A. Way and W. L. White, Spartanburg, S. C.—p. 760.

Use of Polarized Light in Analysis of Calculi and in Study of Crystals in Tissue: Preliminary Report on Method Employed. E. L. Prien, Boston.—p. 765.

Sulfathiazole and Staphylococcus in Urinary Infections. G. Carroll, L. Kappel and B. Lewis, St. Louis.—p. 770.

Effect of Small Doses of Testosterone Propionate on Testis and Accessory Sex Organs. H. S. Rubinstein and A. A. Kurland, Baltimore.—p. 780.

Response of Cardiovascular System to Intravenous Fluids.—Murphy determined the effect of fluids on tests of cardiovascular function in 149 elderly individuals with and 10 without evidence of heart disease. The cardiovascular disturbances were arterial hypertension, valvular heart disease, coronary artery disease or hypertensive heart disease. The author's purpose was to test the tolerance of the cardiovascular system to added strain from increased blood volume and therefore he did not confine the use of fluids strictly to the standards of safe therapeutic control. Records were made of changes in the plasma and blood volume, erythrocyte counts, hematocrit determinations, the amount of plasma protein, hemoglobin, pulse rate, blood pressure, respiratory rate, venous pressure, blood circulation time, the vital capacity changes, the minute volume urinary output and in the electrocardiograms. The rate of administering the fluids varied from 10 to 30 cc. per minute. Clinical observations show that some patients with compensated heart lesions tolerate large quantities of fluid very well whereas others with similar lesions respond badly. The author knows of no test that will enable one to determine the response beforehand. In the presence of an organic defect, regardless of the compensation, small amounts of fluids should be given, preferably isotonic, and they should be given slowly. Patients with pneumonia or other serious pulmonary impairment tolerate large

quantities of fluid badly under all circumstances. The venous pressure is not always sufficiently elevated to be the sole cause of the hemoconcentration occurring in heart failure or the decreased plasma and blood volume changes observed in patients going into failure. Vital capacity likewise cannot be regarded as a test for determining beforehand how the cardiovascular system will respond to fluids, yet it is an important aid in estimating the presence or absence of pulmonary congestion. If the heart is impaired, the speed of giving fluids intravenously is the most important single factor. Most patients will tolerate fluids well if they are given slowly, from 5 to 10 cc. per minute. The ultimate fate and mode of distribution or loss of injected fluid is dependent on the nature of the solution, the state of hydration, the degree of cardiac compensation, the level of the venous pressure and the chemical changes in the blood and tissues that influence osmotic pressure. Patients without heart failure will tolerate fluids in amounts up to 3,000 cc. a day even when dehydration is absent. The main safeguard for guidance in intravenous fluid therapy is the condition of the cardiovascular system. Hematocrit, plasma protein, venous pressure and vital capacity determinations are of great importance in determining the kind and quantity of fluid to be given and the speed of injection, but they do not aid in eliciting the ability of the cardiovascular system to accommodate fluids.

Kentucky Medical Journal, Bowling Green

39:153-196 (May) 1941

Uterine Bleeding. M. J. Henry, Louisville.—p. 157.
Indications for and Choice of Operation in Peptic Ulcer. F. W. Rankin and C. C. Johnston, Lexington.—p. 163.
Relative Value of Insulins in Treatment of Diabetes. C. C. Turner, Glasgow.—p. 170.
Evaluation of Present Status of Male Hormone Therapy. J. R. Hendon, Louisville.—p. 176.
Appendical Peritonitis. W. Barrow, Lexington.—p. 184.
History of the Jefferson County Medical Society. E. L. Heflin, Louisville.—p. 188.

Military Surgeon, Washington, D. C.

88:459-586 (May) 1941

Medical Service at the First Army Maneuvers of 1940, with Special Reference to Work of the First Evacuation Hospital. J. L. Kantor.—p. 459.
Therapeutic Use of Sulfanilamide and Related Compounds. H. K. Mohler.—p. 473.
Newer Concepts in Treatment of Varicose Veins. L. K. Stalker.—p. 486.
Aid to Diagnosis of Latent Syphilis—Luetism. W. S. Bainbridge.—p. 492.
Proposal for Training of Officers of the Medical Department Reserve Corps, U. S. Army, Assigned to the Laboratory Service, Both on Active and Inactive Status. A. G. Hulett.—p. 499.
Headache. W. P. E. Berwald and H. W. Williams.—p. 504.
Veterinary Research and Public Health. R. A. Kelser.—p. 509.
Preoperative Medication for Reduction of Gingival Infection Before Oral Surgery. F. Kaiser.—p. 519.
Some Observations Regarding Arthritis and Allied Conditions. B. W. Harris.—p. 523.
Temporomandibular Joint. C. E. Sheets Jr.—p. 529.
Military Psychiatry. J. A. Brussel.—p. 539.
Extension Hanging Cast for Fractures of Humerus. A. M. Wright, G. A. Koenig and B. Shafiroff.—p. 542.
Fly Control. R. W. Browne.—p. 546.
Compression Fracture of Cervical Vertebrae with Spondylitis Osteoarthritica. E. R. Eaton.—p. 549.
Dr. Samuel Gridley Howe: "A Yankee Cervantes." J. M. Phalen.—p. 553.

New Orleans Medical and Surgical Journal

93:543-600 (May) 1941

Reflections on Edmond Souchon (1841-1924). I. Cohn, New Orleans.—p. 550.
Treatment of Postoperative Adynamic Ileus and Obstruction of Small Bowel with Miller-Abbott Tube. I. W. Kaplan and M. L. Michel, New Orleans.—p. 558.
Fallacies in Treatment of Heart Disease. P. D. White, Boston.—p. 565.
Treatment of Compound Fractures. P. C. Colonna, Oklahoma City.—p. 570.
Nonbloody Vaginal Discharges of Pregnancy. W. F. Guerriero and R. E. Arnell, New Orleans.—p. 574.
Studies with Antigens: X. Statistical Methods for Analysis of Comparative Skin Reactions. M. R. Pabst, C. H. Boatner and B. G. Efron, New Orleans.—p. 579.
Bagassosis: Fungus Disease of Lung: Case Report. S. C. Jamison and J. Hopkins, New Orleans.—p. 580.
Extramedullary Aneurysm of Spinal Cord. D. H. Echols and R. G. Holcombe Jr., New Orleans.—p. 582.

Public Health Reports, Washington, D. C.

56:751-814 (April 11) 1941

Mobile Laboratory Units of the Ohio River Pollution Survey. F. E. DeMartini.—p. 754.
Domestic Water and Dental Caries: II. Study of 2,832 White Children, Aged 12-14 Years, of Eight Suburban Chicago Communities, Including Lactobacillus Acidophilus Studies of 1,761 Children. H. T. Dean, P. Jay, F. A. Arnold Jr. and E. Elvove.—p. 761.
Application of Human Serum Opacity Reaction for Evaluating Antitoxin Binding Power (I.B) of Clostridium Perfringens (Type A) Toxoid. S. C. Seal and Sarah E. Stewart.—p. 792.
Disabling Morbidity Among Industrial Workers, Final Quarter of 1940, with Index of Previous Publications of This Series. W. M. Gafafer.—p. 799.

56:871-928 (April 25) 1941

Recent Developments Relating to Public Health Interest in Housing. J. C. Leukhart.—p. 871.
Prevalence of Poliomyelitis in the United States in 1940. C. C. Dauer.—p. 875.
Preliminary Survey of Anopheline Mosquito Fauna of Southeastern Minnesota and Adjacent Wisconsin Areas. R. H. Daggy, O. J. Muegge and W. A. Riley.—p. 883.
Postsanatorium Tuberculosis Survival Rates in Minnesota. H. E. Hilleboe.—p. 895.
Choriomeningitis Virus Infection Without Central Nervous System Manifestations: Report of Case. C. Armstrong and J. W. Hornibrook.—p. 907.
Further New Species of Ornithodoros from Bats (Acarina: Argasidae). R. A. Cooley and G. M. Kohls.—p. 910.

56:929-984 (May 2) 1941

How Health Departments Function with Respect to Specific Problems. J. O. Dean and Evelyn Flook.—p. 929.
Bactericidal Effect of Paraffining of Paper Board Used for Paper Milk Containers. F. J. Moss, R. C. Thomas and Mildred K. Havens.—p. 944.
Dick Reaction and Scarlet Fever Morbidity Following Injections of Purified and Tannic Acid Precipitated Erythrogenic Toxin. M. V. Veldee, E. C. Peck, J. P. Franklin and H. R. DuPuy.—p. 957.

56:985-1038 (May 9) 1941

Special Problems in Our Health Defenses. P. V. McNutt.—p. 988.
"Sporadic" Poliomyelitis, with Special Reference to Geographic and Chronologic Distribution in Tennessee in the Eighteen Months Ended June 30, 1940. L. L. Lumsden.—p. 992.
Clinical Study of Poliomyelitis in Charleston County, South Carolina, 1939. D. J. Davis, F. J. Weber and Margaret S. Arey.—p. 1007.
Causes of Physical Disqualification Under the Selective Service Law: Early Indications. R. H. Britten and G. St. J. Perrott.—p. 1017.
Portable Unit for Determination of Halogenated Hydrocarbons. H. C. Dudley.—p. 1021.

Rocky Mountain Medical Journal, Denver

38:337-416 (May) 1941

Metabolic Significance of Vitamins. A. H. Smith, Detroit.—p. 354.
Malunited and Ununited Fractures of Both Bones of Forearm. R. K. Ghormley, Rochester, Minn.—p. 358.
Prolonged Labor. E. L. King, New Orleans.—p. 363.
*Induced Anoxemia in Diagnosis of Coronary Artery Disease: Investigation as to Its Value. C. T. Burnett, M. G. Nims and C. J. Josephson, Denver.—p. 367.
Neurosyphilis: Its Industrial Implications. P. Work, Denver.—p. 371.
Utilization of Mental Faculties as Aid in Treatment of Disease. D. E. Johnson, Alamosa, Colo.—p. 375.
Expansion of Blue Cross Plan to Serve Additional Communities. W. S. McNary, Denver.—p. 380.

Induced Anoxemia in Coronary Disease.—Burnett and his colleagues studied the effects of induced anoxemia in 50 normal persons from 20 to 30 years of age. In general their observations have substantiated those reported by Levy. While the appearance of angina-like pain is important, the interruption of the test depends more on the electrocardiographic changes which may occur under conditions of induced anoxemia. The electrocardiogram of the normal individual subjected to this test undergoes slight changes in the RS-T intervals and T wave amplitude. According to Levy, in the presence of coronary insufficiency these RS-T changes become greatly exaggerated. Partial or complete reversal of T waves may occur with or without these RS-T deviations. These changes are promptly reversible by inhalation of pure oxygen. Their demonstration may prove to be of some value in the diagnosis of insufficiency of the coronary circulation. Carelessly applied, the test is hazardous, but under meticulous conditions it is not dangerous and when carried out by experienced workers is of diagnostic value.

Surgery, St. Louis

9:493-656 (April) 1941

Comparative Results in Use of Living and Preserved Fascia as Suture Material in Bone. A. R. Koontz and R. T. Shackelford, Baltimore.—p. 493.

Studies on Shock: III. Variability of Shock Syndrome in Toxic Drug Shock. F. Neuwelt, S. O. Levinson and H. Necheles, Chicago.—p. 503.

*Staphylococcic Septicemia: Treatment by Chemotherapy and Serotherapy. S. L. Goldberg and E. D. Bloomenthal, Chicago.—p. 508.

Inefficacy of Lumbar Puncture for Removal of Red Blood Cells from Cerebrospinal Fluid. J. M. Meredith, University, Va.—p. 524.

Absorption of Sulfanilamide from Burned Surfaces. D. H. Hooker and C. R. Lam, Detroit.—p. 534.

Peptic Ulcer and Diarrhea Following Removal of Prevertebral Ganglions in Dogs: Antispasmodic Effects of Magnesium Sulfate, Pentobarbital and Atropine Sulfate. R. Lium, Portsmouth, N. H.—p. 538.

*Large Islet Cell Tumor of Pancreas. A. Brunschwig, Chicago.—p. 554.

Strangulated Hernia Reduced En Masse. D. Casten and M. Bodenheimer, New York.—p. 561.

Acute Complete Obstruction of Hepatic Veins: Report of Case Simulating Acute Abdomen. A. M. Dickinson, Albany, N. Y.—p. 567.

Syphilis of Stomach Necessitating Total Gastrectomy. C. H. Avent, Memphis, Tenn.—p. 571.

*Epiloia: Report of Cases. L. M. Quill and Esther C. Marting, Cincinnati.—p. 581.

System of Surgical Follow-Up and Record Keeping. C. E. Gardner Jr., Durham, N. C.—p. 591.

*Successful Ligation of Patent Ductus Arteriosus. C. J. Miangolarra and E. Hull, New Orleans.—p. 597.

Staphylococcic Septicemia.—Goldberg and Bloomenthal review instances of staphylococcic septicemia treated at the Michael Reese Hospital from 1934 through 1939. The series includes 32 cases of staphylococcemia, 19 males and 13 females, with 14 deaths. The diagnosis in each was established by recovery of the organism from the circulating blood stream in pure culture at least once. There were 26 instances of Staphylococcus aureus and 6 of Staphylococcus albus. In 23 patients the organism was also recovered from the primary or metastatic focus or both. The organisms entered the body through various ports, most frequently through the skin. The initial cutaneous lesion can be so small as to evade the most careful search. In 9 the initial focus was recovered as osteomyelitis; these may have been of such an origin. In 14 others the cutaneous focus was known; 11 carbuncles or furuncles, 1 infected bulla, 1 laceration and 1 infection secondary to trichophytosis of the feet. Infection of the upper part of the respiratory tract preceded the bacteremia of 6 patients, in 1 the origin was puerperal sepsis, in 1 infection of the urinary tract and in 1 a postoperative infection of a nasoplasty incision. The time between the appearance of the initial focus of infection and blood stream invasion varied from forty-eight hours to several weeks. Before the advent of sulfanilamide general treatment was mainly supportive and local treatment consisted of rest, heat, incision and drainage of collections of pus. Sulfanilamide alone was used for 6 patients, all of whom died. Six other patients were treated with sulfanilamide in combination with staphylococcus antitoxin, and 4 of these recovered. It did not appear that sulfanilamide had any specific value, as the clinical course of most of these patients was either moderately severe or protracted and recovery without the use of specific therapy was expected. Fifteen patients, 4 of whom were more than 14 years old, were treated with staphylococcus antitoxin alone or in combination with sulfanilamide or sulfapyridine. The total mortality rate was 33⅓ per cent. There were only 3 deaths among the 11 younger patients. The authors' experience, particularly in the more recent cases, in which larger doses of antitoxin (100,000 to 180,000 units) were administered, leads them to believe that antitoxin is of definite value and that it should be administered early and in sufficient dosage. The 6 patients admitted to the hospital during 1939 were treated with sulfapyridine. In 3 it was combined with staphylococcus antitoxin. All 6 patients recovered. The initial dose of the drug was approximately 1½ grains (0.1 Gm.) per pound of body weight for each twenty-four hours. The sulfapyridine blood level should be at least 6 mg. per hundred cubic centimeters, preferably between 6 and 9 mg. With the subsidence of symptoms, the dose should be reduced gradually. In all 6 patients, and especially the 3 patients also given staphylococcus antitoxin, the clinical improvement was definite and rapid and occurred soon after treatment was instituted. Sound surgical treatment of local lesions is paramount. If the infection is in soft tissues, it should be treated conservatively and not manipulated until pus is formed. Heat and possibly roentgen irradiation are of value in localizing the infection. Collections of pus should be evacuated. Chemotherapy and serotherapy appear synergistic in action, and both agents should be used in staphylococcic septicemia. Early clinical diagnosis and prompt institution of therapy before the diagnosis is corroborated by blood culture is important, as are supportive treatment and sound surgical measures.

Islet Cell Tumor of Pancreas.—Brunschwig reports a case of an islet cell tumor of the pancreas of unusual size. The tumor enlarged from about 7 cm. in diameter to 15 cm. in four months without gross evidence of metastases. This increase was not accompanied by a parallel increase in the number or severity of attacks, as judicious spacing of food intake controlled them fairly well. The patient's postoperative diabetes is accounted for on the basis of the relative inactivity of the remaining normal islet tissue as the result of the large output of insulin from the tumor and of the fact that, when the latter was suddenly withdrawn, the remaining normal pancreas could not cope with the sudden increased demands on it. Recovery of adequate function (insulin production) occurred after three weeks of injections of insulin. A study of the sections warrants the diagnosis of malignant neoplasm on morphologic criteria alone. However, from the biologic standpoint the absence of metastases while the primary tumor grew so rapidly in a relatively young adult (32 years of age) favors the view that the process was probably benign at operation or at least still localized. Previous experience has shown that, while microscopically these tumors might appear malignant, their removal has resulted in prolonged survival without recurrent attacks of hyperinsulinism. At the first exploration two tumors were observed. These apparently had fused and formed the large tumor removed at the second operation. While the smaller tumor might have been a local metastasis, it is equally probable that at the onset there were two distinct neoplasms arising from different islets.

Epiloia.—Quill and Marting describe the syndrome of epiloia observed in 7 persons of three generations. Although the mother of the first and seventh patients (the grandmother of the other 5 patients) was not examined clinically, the history of epileptic seizures, adenoma sebaceum and nailbed tumors makes the diagnosis of epiloia a valid presumption. If this is granted the study reveals direct passage of the disease through three generations. The study discloses that, if the epiloia patient lives through puberty and is capable of mating, transmission of the disease is possible. The reason for so few examples of transmission lies in the fact that the average affected patient is so mentally and physically incapacitated at puberty that marriage and mating do not occur. Study of the family tree discloses that 1 daughter (case 1) of the original epiloia patient transmitted the disease in all its intensity to all 4 of her children who lived to puberty; whereas, the other daughter (case 7) with signs of the disease apparently did not pass it on to her 2 older children. There is no evidence of the disease in the other 2 children of the original epiloia patient. Neither was it found in their children. This suggests that the disease is not recessive but rather incompletely dominant. The nailbed tumors, usually an uncommon accompaniment, are of particular interest because of their presence in all 7 cases. The presumption here is that epiloia is transmitted even in its minute details. The direct transmission of the disease from parent to offspring through three generations proves that the syndrome is hereditary, i. e. genotypical as suggested by Fabing.

Successful Ligation of Patent Ductus Arteriosus.—Miangolarra and Hull cite successful ligation of a patent ductus arteriosus in a 20 year old girl by the transpleural technic of Gross. They feel that all such operations should be recorded in order that proper evaluation may be placed on the results of the procedure. Seven months after the operation the patient's status is approximately the same except that there is no evidence of patency of the duct. The patient may be spared the later development of heart failure, vegetative endarteritis or other complications of this anomaly.

Tennessee State Medical Assn. Journal, Nashville

34:83-120 (March) 1941

Pseudosinusitis. E. Orr, Nashville.—p. 83.
The Place of Vitamins in Therapy. J. B. Youmans, Nashville.—p. 88.
Treatment of Appendical Peritonitis. W. D. Haggard and J. A. Kirtley Jr., Nashville.—p. 97.

34:121-160 (April) 1941

Seven Correctable Conditions Occasionally Labeled Idiopathic Epilepsy. T. F. Frist, Nashville.—p. 121.
Choice of Anesthesia in Acute Surgical Emergencies. H. M. Ausherman, Chattanooga.—p. 128.
Diagnosis and Management of Thyroid Disease. H. Wilson, Memphis. —p. 135.
The East Tennessee Medical Association. W. T. Mathes, Greeneville.— p. 138.
Management of Reactions to Arsenicals. C. Shaw, Chattanooga.—p. 142.

Texas State Journal of Medicine, Fort Worth

36:771-854 (April) 1941

Consideration of Some Common Pediatric Problems. A. Brown, Toronto. —p. 778.
*Tumors of Salivary Glands. A. O. Singleton and N. Duren, Galveston. —p. 784.
Uterine Endometriosis. T. A. Pressly, San Antonio.—p. 792.
Benign Lesions of Duodenum. R. T. Wilson, Austin.—p. 797.
Roentgenologic Diagnosis of Diseases of Stomach. L. M. Garrett, Corpus Christi.—p. 799.
*Ophthalmoplegic Migraine. R. K. Daily, Houston.—p. 802.
Public Health and the Practice of Medicine. W. C. Williams, Nashville, Tenn.—p. 806.

37:1-72 (May) 1941

Technic and Value of Colposcopy. Clara K. Duncan, Houston.—p. 6.
Progress in Surgery of Large Bowel. G. W. Waldron, Houston.—p. 13.
"Cold Punch" Type of Prostatic Resection. J. H. Shane, Dallas.— p. 17.
Survey of the Present Status and Problems of Sterility. F. Cone, Houston.—p. 20.
Resection of Presacral Nerve for Dysmenorrhea and Pelvic Pain. J. W Hendrick, Amarillo.—p. 26.
Construction of Artificial Vagina. J. A. Heymann, Wichita Falls.— p. 30.
Common Skin Diseases of Childhood. L. A. Brunsting, Rochester, Minn. —p. 33.
Regulation of Blood Sugar in Diabetes by Sublingual Administration of Insulin. D. R. Sacks, San Antonio.—p. 39.
Methods of Hemorrhage Control in Nose and Throat. K. Gill, Corpus Christi.—p. 43.
Bacon Saunders. K. H. Aynesworth, Waco.—p. 47.
Our Public Health Status. G. W. Cox, Austin.—p. 53.

Tumors of Salivary Glands.—Singleton and Duren report 55 tumors arising in the salivary glands and related tissues; 40 were mixed tumors and 15 carcinomas. The race, sex and site (side of the face) were approximately equal. The average age for the series was 38 years, for the mixed tumors 31.5 years and for the malignant tumors 52.3 years. The preoperative duration of the mixed tumors averaged five and four-tenths and that of the malignant tumors one and twenty-four one hundredths years. Recurrence data are available for 41 cases, 30 of which were mixed tumors and 10 recurred. Ten of the 15 malignant tumors were primarily excised at the John Sealy Hospital; 6 of the patients died from progression of the malignant process, and 3 of the 4 patients now alive have been free from recurrence for approximately one year. The fourth has been free from recurrence for four months. Regional metastases were present in 11 cases and general metastases in 1. The death of 9 patients was directly attributable to the tumor. The authors believe that, if the morbidity and deformity in addition to the mortality are considered, a more radical excision seems justified. They state that the tumors so treated tended to recur less often than those not so widely excised. In contrast to some authors' experience the smaller tumors, when excised, tended to recur less often than the larger ones.

Ophthalmoplegic Migraine.—Ophthalmoplegic migraine, according to Daily, describes a clinical syndrome characterized by attacks of severe headache and paralysis of the ocular muscles, with ptosis as a frequent symptom. A severe headache localized to one side develops in the course of the attack. It starts, as a rule, at the temple and spreads to the occipital and frontal regions. The headache is of the constant dull, harrowing type, as occurs in migraine and tumors of the brain. It is accompanied by nausea and vomiting but not by scintillating scotoma or any of the visual sensations common to migraine. Nausea accompanies the headache but may precede it or follow. After from one day to a week of headache, paralysis of the oculomotor muscles sets in on the side of the pain. As the paralysis develops the headache and nausea abate, but there are cases in which this order is reversed. The duration of pain and paralysis and its extent and intensity are subject to wide variations in individuals and in attacks. It does not seem logical that a case of persistent paralysis and a case of transitory paralysis should be caused by the same pathologic process. Two types may be considered: the persistent paralysis, which must remain under the suspicion of an organic lesion, and transitory paralysis, which, the author suggests, might be considered as a vasomotor functional neurosis of the posterior cerebral artery. If the process is a transitory angiospasm the result is migraine with the various striking visual sensory phenomena. If the process is a vasodilatation with edema the result is a compression of the oculomotor nerves and paralysis. The intensity of the storm is obviously greater in ophthalmoplegic migraine; the shortest attack lasts much longer than the severest attack of ordinary migraine. What factor directs the vascular disturbance into a spasm or dilatation is not known. That psychic and emotional disturbances are important exciting factors has been established. The differential diagnosis has to eliminate intracranial tumors, aneurysm of the vessels at the base of the brain, congestion of the cavernous sinus, syphilis, tabes, syringomyelia, chronic meningitis, multiple neuritis, chronic ophthalmoplegia, chronic and subacute ophthalmoplegia combined with bulbar disease and disease of the anterior horn, asthenic ophthalmoplegia, multiple sclerosis and hysteria. Treatment is that of a functional neurosis and the prognosis is good. With a better understanding of the pathologic changes and therapy of functional neurosis, recurrent ophthalmoplegic migraine should be eliminated.

Virginia Medical Monthly, Richmond

68:247-312 (May) 1941

Peripheral Vascular Disease. D. G. Chapman, Richmond.—p. 247.
Fibroepithelial Tumors, Chronic Cystic Mastitis and Carcinoma of Breast. E. R. Whitmore, Washington, D. C.—p. 251.
Management of Thyroglossal Cyst: Report of Ten Cases. E. G. Gill, Roanoke.—p. 267.
Aneurysm of Abdominal Aorta: Report of Case of Rupture into Duodenum. H. M. Smith Jr., Philadelphia.—p. 270.
Sulfathiazole in Infections of Urinary Tract and in General Septicemia: Report of Fifty Cases. W. C. Stirling, Washington, D. C.—p. 271.
Lithopedion of Seven Years' Duration Followed by Hydatiform Mole: Case Report. I. Tractenberg and W. Oliver, Brooklyn.—p. 277.
Sulfanilamide Therapy in Madura Foot. J. M. Dixon, Roanoke.—p. 281.
Habit. R. Blankinship, Richmond.—p. 282.
Four Problems Concerning Adenomatous Goiter. R. S. Anderson, Rocky Mount, N. C.—p. 285.
The Pediatrician's Role in Mental Hygiene. B. B. Jones, Richmond.— p. 286.

War Medicine, Chicago

1:301-462 (May) 1941

The Navy's Medical Department. W. J. C. Agnew and N. L. Saunders, Washington, D. C.—p. 301.
Plan for Collection, Transportation and Administration of Whole Blood and Plasma in Warfare. E. L. DeGowin and R. C. Hardin, Iowa City.—p. 326.
Effective Technic for Desiccating Plasma in Useful Quantities: Sterile Unit Desiccator. T. R. Folsom, New York.—p. 342.
What Physicians Expect from Psychiatry. E. C. Cutler, Boston.— p. 352.
Psychiatric Factors in the Medical Examination. J. H. Pratt, Boston. —p. 358.
Military Psychiatry and the Selective Service. W. C. Porter, Washington, D. C.—p. 364.
Economic Aspect of Psychiatric Examination of Registrants. M. Cooley, Washington, D. C.—p. 372.
Peculiar Personalities: Disorders of Mood; Psychopathic Personality. R. W. Hall, Washington, D. C.—p. 383.
Study of Specific Data in Lives of 183 Veterans Admitted to St. Elizabeths Hospital. A. Simon, Margaret Hagan and R. W. Hall, Washington, D. C.—p. 387.
Disorders with Structural Features. A. Simon, Washington, D. C.— p. 392.
Psychosomatic Disorders. A. Myerson, Boston.—p. 404.
Schizoid and Related Personalities. D. A. Thom, Boston.—p. 410.
Neuropsychiatric Examination of Applicants for Voluntary Enlistment and Selectees for Induction. Circular Letter No. 19.—p. 418.
Active Immunization Against Tetanus by Vaccination with Tetanus Toxoid. Circular Letter No. 34.—p. 426.
Storage of Mapharsen. Form Letter.—p. 429.
Directions for Use of Yellow Fever Vaccine in Immunization Against Yellow Fever. Form Letter.—p. 429.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Journal of Children's Diseases, London

38:1-58 (Jan.-March) 1941

Purpura in Course of Measles: Case Treated with Vitamin P. A. A. Miller.—p. 1.
Anatomy of Lactating Breast. S. Engel.—p. 14.

British Journal of Radiology, London

14:147-186 (May) 1941

Inverse Square Law Errors in Gamma Ray Dose Measurements. F. W. Spiers.—p. 147.
Chloroma. T. A. Kemp and E. R. Williams.—p. 157.
Spondylolisthesis Affecting Fourth Lumbar Vertebra. W. M. E. Topping.—p. 162.
Regional Ileitis. G. Friedlaender.—p. 164.
X-Ray Examination in Relation to Treatment of Air Raid Casualties. F. Samuel.—p. 171.
Near Distance Therapy. F. Roberts.—p. 175.
Some Radiologic Observations on Postcricoid Obstruction and Anemia. A. S. Johnstone.—p. 177.
Research into Physical Factors Concerned in Indirect Radiography: Paper II. Light Transmission of X-Ray Protective Lead Glass. R. Herz and B. Stanford.—p. 181.

British Medical Journal, London

1:469-500 (March 29) 1941

*Use of Glycerin-Sulfonamide Paste ("Euglamide") in Treatment of Burns. J. M. Robson and A. B. Wallace.—p. 469.
Digestive Disorders in the Forces. J. G. Graham and J. D. O. Kerr.—p. 473.
Experiences with Sulfanilamide Derivatives in Some Tropical Conditions. K. V. Earle.—p. 476.
Laboratory Infection with Brucella Abortus. H. N. Green.—p. 478.
Convulsion During Nonconvulsive Faradic Shock Therapy. G. Caplan.—p. 479.

Glycerin-Sulfonamide Paste for Burns.—Robson and Wallace outline a method of treating burns of the hands which overcomes the disadvantages of tanning methods. The edema and sepsis under a rigid tan, together with prolonged immobilization, have played their part in limiting movement and causing contracture and, in extreme cases, ischemic necrosis. The principle of the method is the application to the burn of a water soluble sulfonamide in glycerin. The sulfonamide compound (albucid soluble) chosen was known to be innocuous to tissues. As glycerin by itself is unsuitable for surface application, the albucid solution was converted into a paste by adding a suitable quantity of kaolin. The paste produces effects usually associated with cataplasma kaolin, an increase in the flow of lymph containing antibodies and white blood corpuscles which increase the local defense mechanism. To prevent the paste from drying, a small quantity of cod liver oil is incorporated. This may have additional advantages. The treatment of burns with this paste allows early movement. For preparing the paste 5 Gm. of soluble albucid powder is mixed with 100 cc. of glycerin; the mixture is heated cautiously and stirred. While the mixture is still warm, 10 cc. of cod liver oil is added and thoroughly stirred in. This solution is mixed with about 80 Gm. of fine kaolin until a smooth permanent paste is produced. The paste has been used with success in second and third degree burns of the hand, face, scalp and buttocks, in second degree burns of the penis and scrotum, in septic burns following twenty-four hours of wet dressings, and for not extensive second degree burns. An anesthetic may be required for cleansing. Before cleansing or applying the paste, cultures are made from the surface of the burn and from blister fluid. In second degree lesions the affected part is cleansed with gauze wrung out of isotonic saline solution at body temperature. Blisters and loose epidermis are snipped and removed. In third degree burns the surface is gently washed with warm physiologic solution of sodium chloride and all foreign material is removed. The paste is applied thickly on sterile white lint, gauze or linen; for burns of the face, a mask is cut out and put over the burned area. Further dressing is applied if necessary, and the whole is covered with wool and bandaged. In burns of the face and flexure regions the paste and dressings are reapplied daily, but in other parts they may be kept on for three days before being changed. When infection is present it is essential to maintain a high local concentration of the sulfonamide, and the paste is reapplied daily. At each change of dressing, the affected part is wiped clean with wool pledgets soaked in physiologic solution of sodium chloride or cod liver oil. Most of the burns treated have been of the second degree and have occurred in children. Neither the interval between the injury and admission to the hospital nor the nature of the first aid treatment appeared to influence the ultimate result. The hands, face, abdomen, buttocks, feet, penis and scrotum responded well, whereas the axilla and shoulder region showed less response but nevertheless did well. Pain was experienced by a few patients on application of the paste; irritation was absent. As a rule an early healing surface was obtained and therefore patients were discharged earlier from the hospital. The rate of healing with the paste ("euglamide") compares favorably with that obtained with tannic acid. Satisfactory results were obtained in facial burns. The method allows a certain amount of freedom which cannot be permitted if tannic acid has been used. The results of treating a few burns which involved the whole skin have been promising. The paste has also proved of value in burns which had become septic after coagulating agents were used. In such cases euglamide was applied to the granulating surface after the coagulum was removed and healing was rapid.

Edinburgh Medical Journal

48:289-360 (May) 1941

Action of Drugs on Human Uterus. J. A. Gunn.—p. 289.
Some Comments on Present State of Cancer Theory. J. P. McGowan.—p. 305.
Note on Use of Sulfapyridine in Pneumonia of Children. R. M. Murray-Lyon and R. W. Biagi.—p. 313.
Use of Oxalated Finger Blood for Routine Hematologic Examination. S. Hay.—p. 317.
Production of Glomerulonephritis in Animals by Injection of Serum from Patients Suffering from Nephritis. B. Schober.—p. 322.
The Lighter Side of Edinburgh Medical Life at the End of the Nineteenth Century. A. C. Brown.—p. 335.

Journal of Neurology and Psychiatry, London

4:1-96 (Jan.) 1941

Cerebral Vascular Lesions and Tentorial Pressure Cone. R. S. Allison and J. E. Morison.—p. 1.
Mechanism of Contrecoup Injury. A. F. Goggio.—p. 11.
Head Injuries and Meningitis. E. A. Linell and W. L. Robinson.—p. 23.
Effect of Choline-like Substances on Cerebral Electrical Discharges in Epilepsy. D. Williams.—p. 32.
Critical Review: Dementia in Middle Age. W. H. McMenemey.—p. 48.

Journal of Pathology and Bacteriology, Edinburgh

52:155-282 (March) 1941

Evolution of Carcinoma of Mamma. R. Muir.—p. 155.
Squamous Metaplasia of Columnar Epithelium in Human Cervix. R. Carmichael and B. L. Jeaffreson.—p. 173.
*Active Immunization Against Gas Gangrene and Tetanus. W. J. Penfold, J. C. Tolhurst and D. Wilson.—p. 187.
Autohemagglutination and Anti-O Agglutinins. G. C. Dockeray and H. Sachs.—p. 203.
Benign Cerebral Telangiectasis: Two Cases. W. Blackwood.—p. 209.
Some Developmental Abnormalities of Thymus and Parathyroids. J. R. Gilmour.—p. 213.
Isolation of Cholesterol from Oily Droplets Found in Association with the L 1 Organism Separated from Streptobacillus Moniliformis. S. M. Partridge and Emmy Klieneberger.—p. 219.
Some Observations on Healing of Experimental Wounds in Skin of Rabbit. J. S. Young, J. A. Fisher and M. Young.—p. 225.
Action of Certain Sulfonamide Drugs on Growth of Bacteria in Urine. J. W. Bigger and P. A. McNally.—p. 247.

Active Immunization Against Gas Gangrene and Tetanus.—Experience with active immunization of 12 volunteers with Bacillus welchii (type A) alum precipitated toxoid suggested to Penfold, Tolhurst and Wilson its use as prophylaxis against gas gangrene in the army. The volunteers received a total of thirty-three injections. There were only local reactions and these were mild and possibly due to traces of formic acid in the formaldehyde used to prepare the toxoids. All the volunteers responded with the production of antitoxin in their serums and the serums injected into mice protected the animals against living cultures of B. welchii. The maximal response of the volunteers following either two or three injections varied from $\frac{1}{20}$ to $\frac{1}{2}$ unit of antitoxin per cubic centimeter of serum.

The courses of immunization varied with the volume of the injections, the intervals between them and the batch of alum precipitated toxoid used. After two or three injections, antitoxin was detectable for at least five months in the serums of 9 volunteers. One of these had antitoxin for at least two and a half years; in 3 others none was detectable after eighteen months. Of the remaining 3 persons, 1 showed antitoxin after two months (his serum was not tested at a later date), 1 had antitoxin after two months but not after fourteen months and 1 had less than $\frac{1}{30}$ unit after three months, when a further injection was given. Three other persons had an extra injection after nine, five and five months. In each instance there was a sharp rise in the antitoxic titer.

Lancet, London

1:437-470 (April 5) 1941

Effort Syndrome: Diagnostic and Therapeutic Value of Exercises. A. Abrahams.—p. 437.
*Prognosis of Acute Intestinal Obstruction: Experiments with Intraperitoneal Sulfanilamide. R. V. Hudson, R. Smith and F. R. Selbie.—p. 438.
Preservation of Blood for Sugar Analysis. R. D. Lawrence.—p. 442.
*Treatment of Bacterial Endocarditis with Heparin and Sulfapyridine. S. Sevitt.—p. 443.
Failure of Heparin in Subacute Bacterial Endocarditis. C. M. Fletcher.—p. 444.
Calcification in Muscles of Forearm. R. Shackman and C. J. Longland.—p. 445.
Epanutin and Electric Convulsion Therapy. R. E. Hemphill and W. G. Walter.—p. 446.

Prognosis of Acute Intestinal Obstruction.—Hudson and his associates studied a consecutive series of 670 cases of abdominal obstruction. In 62 a segment of the intestine was not considered viable and a resection was carried out; 48 of these patients died, giving a fatality rate of 77.4 per cent. Of 300 patients with strangulated external hernia 30 required resection; 24 of these died, a fatality rate of 80 per cent. Of the remaining 270 patients having simple release only 18 died, giving a fatality rate of 6.7 per cent. In trying to determine the reason for this significant difference in the prognosis when the intestine changes from viable to nonviable, the authors find that the majority survive the immediate hazards of operation and the next few days. They may die at any time up to six weeks after operation, the most dangerous periods being about the fifth and the tenth day. The symptoms are typical of an increasing mechanical obstruction—colicky pains, exaggerated peristalsis, vomiting and increasing distention. At necropsy the cause of death is found to be a plastic peritonitis of a variable degree, with much adhesion of intestine to intestine and intestine to abdominal wall, and an edematous intestine full of foul material, with multiple sites of obstruction. The author believes that some organisms probably pass through the intestinal wall before operation but not many unless gangrene and perforation are present; the main contamination of the peritoneum probably takes place at operation and is due to the intestine being opened. If the 670 patients are placed in two groups, according to whether the intestine was opened or not, there are 224 whose intestine was opened, of whom 137 died; but of 446 having only simple release 23 died. The prognosis for patients requiring opening of the intestine was necessarily much worse, but the difference is so great as to suggest that opening the intestine provides a new lethal factor sufficient to weigh the scales against the patient. Reduction in the deaths can be attained by earlier diagnosis and by more successful treatment and prevention of the lethal complications which result from delay in diagnosis. With a view to preventing such complications, experiments with intraperitoneal sulfanilamide in rabbits demonstrate that (1) sulfanilamide introduced into the peritoneal cavity was absorbed and caused no local inflammation, (2) contamination of the peritoneal cavity with contents of the cecum produced a plastic peritonitis with the formation of abscesses and adhesions and sometimes general peritonitis, (3) this plastic peritonitis could be prevented by introducing sulfanilamide into the peritoneal cavity at the time of the contamination but not by a single large dose of the drug orally, (4) Bacillus coli peritonitis followed resection of intestine with great regularity and (5) prophylactic use of intraperitoneal sulfanilamide usually reduced and sometimes eliminated the inflammatory process, but (6) in the presence of a fulminating infection intraperitoneal sulfanilamide was valueless. So far 2 patients having intestinal resection for acute intestinal obstruction have been treated with sulfanilamide intraperitoneally. In neither of them was there any local or general postoperative obstruction or peritonitis.

Heparin and Sulfapyridine for Bacterial Endocarditis.—Sevitt presents histories of 2 patients treated with heparin and sulfapyridine for bacterial endocarditis. Both died from embolic complications. In one the emboli were pulmonary and in the other cerebral. No organisms were seen microscopically on the vegetations of the first patient. Neither patient underwent a full ten to fourteen day course of treatment; had this been possible the results might have been different. The treatment is undoubtedly highly dangerous; similar cerebral accidents have been reported by Kelson and White and by Witts and Fletcher. However, the treatment seems justifiable for what is invariably a fatal disease.

South African Medical Journal, Cape Town

15:121-140 (April 12) 1941

Mongol Spot in the Cape Colored. S. Matus.—p. 121.
Vaccination Against Epidemic Typhus in South Africa. M. H. Finlayson and G. M. Grobler.—p. 125.
Sporotrichosis Among Miners on the Witwatersrand Gold Mines. L. F. Dangerfield and J. Gear.—p. 128.
Acute Thrombophlebitis of Unknown Etiology. A. C. Fisher.—p. 131.
*Sulfapyridine as Prophylactic Against Cerebrospinal Meningitis. F. C. Gray and J. Gear.—p. 139.

Sulfapyridine Prophylactic of Cerebrospinal Meningitis.—According to Gray and Gear, a bacteriologic investigation of an outbreak of cerebrospinal meningitis in a military camp in Natal revealed approximately 22 per cent of carriers among those examined, making conditions favorable for a widespread epidemic. As sulfapyridine is known to sterilize the cerebrospinal fluid of patients suffering from meningococcic meningitis within three days, it was thought that it might be equally effective in destroying the organisms in the nasopharynx of carriers. If this could be achieved a sudden diminution of the carrier rate would be expected and there would be an interval during which no further cases would occur. The carrier rate was determined on a sample population of the most severely affected battalion. Then all men of this battalion and carriers found in other units were given two tablets of sulfapyridine three times a day for two days. After one day the carrier rate among the sample population was again determined. As a control the carrier rate among an untreated battalion was also determined. Although 70 treated and only 43 untreated men were examined, no carriers were found among the treated but 10 among the untreated. Of still greater significance is the fact that 17 of the treated men were previously proved to be carriers. Under normal conditions, according to Flack (1917), about 20 per cent of contact carriers are infective for less than two weeks and the remainder for periods up to and for longer than twelve weeks. The carriers whose swabs yield a profuse growth of meningococci tend to be more persistent in their infectiveness. The majority of the carriers that the authors treated were of this type and the results of their bacteriologic investigation have to a certain extent been confirmed by subsequent clinical observations and experience. In the month that has elapsed only 1 case of meningitis has occurred in the battalion treated, an officer who did not receive the treatment contracting the disease. A workman in the lines of this battalion who did not receive the prophylactic treatment died of meningitis. During this period several cases of meningitis occurred among the men of the control battalion until they too received the prescribed course of sulfapyridine. On the basis of their investigations Gray and Gear conclude that mass dosage is an efficient means of controlling an epidemic of cerebrospinal meningitis immediately by reducing the carrier rate to negligible proportions and preventing the onset of the disease in those in whom incubation of the infection occurs. The effect of the drug alone cannot be permanent, but they believe that by taking ordinary hygienic precautions the chances of a recrudescence of the disease in epidemic proportions is remote. They recommend that in the event of a case of cerebrospinal meningitis occurring in a semipermanent military camp the carrier rate should be determined if possible, and if it exceeds 20 per cent mass dosing with sulfapyridine is justified. The ordinary hygienic precautions must not be neglected.

Gazzetta degli Ospedali e delle Cliniche, Milan

62:1-20 (Jan. 5) 1941. **Partial Index**

*Cultures of Bone Marrow Obtained by Sternal Puncture in Diagnosis of Bubonic Plague. R. Modica.—p. 11.

Sternal Bone Marrow Cultures in Bubonic Plague.—According to Modica the various methods for identifying Pasteurella pestis are inconvenient because of time consumed. The author made the usual laboratory tests in 12 cases of plague and cultures of bone marrow obtained by puncture. Microscopic examinations of sputum and of material taken from lymph nodes and cultures from the tissue fluids of the lymph nodes cannot be performed in septicemic types of the infection because adenitis and disorders of the respiratory tract appear late in the course of the disease or do not appear at all. The infection was of the general septicemia type in 3 of the author's cases. Positive results were obtained by microscopic examination of the sputum or of the material from involved lymph nodes in 3 of the 9 cases, by culture of tissue fluid of the enlarged lymph nodes in 1 out of 8, by the biologic test in 7 out of 8, by blood culture in 3, and by bone marrow culture in 10 out of 12. The method of bone marrow culture is simple, harmless and valuable for a rapid diagnosis of the bubonic plague. The method has the same degree of sensitivity as the biologic test, without however the inconvenience of the latter. The test can be applied to patients in any stage and with any of the clinical types of the infection. It is of greater diagnostic value than any other diagnostic test.

Policlinico, Rome

48:193-240 (Feb. 3) 1941. **Practical Section**

*Night Blindness in Diseases of Liver. G. Bajardi and A. Galeone.—p. 193.
*Prevention and Therapy of Diphtheritic Paralysis. F. Recchia.—p. 204.

Night Blindness in Diseases of Liver.—Bajardi and Galeone made photometric determinations of the power of adaptation to darkness in 25 patients with disease of the liver and without ocular disorders. The power of adaptation to darkness was diminished in 16 cases. The diminution was proportional to the degree of involvement of the liver parenchyma and stroma and also to the course of the disease, as shown by the clinical symptoms and the results of laboratory tests. Night blindness was present in cases of acute hepatic disease, especially in Laënnec's cirrhosis. It was related neither to the type of the disease nor to jaundice but depended rather on the acuteness of the disease, the course of which it paralleled. The author found hypovitaminosis A in another group of 18 patients with hepatic disease and night blindness. The latter was proportional to the acuteness of hypovitaminosis and it improved on parenteral administration of vitamin A. The authors conclude that changes in the power of adaptation to darkness show more or less acute dysfunction of the liver. The test is of no value for the diagnosis of the type of disease of the liver. The visual disorder is not caused by the disease itself nor by jaundice. It is caused by hypovitaminosis A, the acuteness of which it parallels. It improves on vitamin A therapy.

Prevention and Therapy of Diphtheritic Paralysis.—Recchia reports a statistical study of 3,990 cases of diphtheria observed in the clinic for infectious diseases in Rome. Thirty-four patients who had had no treatment during the acute stage or who received it late in the course of the disease, or in insufficient doses, entered the clinic with paralysis already established. Paralysis developed within five to ten days in 7 cases, within thirteen to thirty-seven days in 20 cases and within fourteen to fifty-nine days in 7 cases. Antiserum was administered to 3,956 patients during the acute phase of diphtheria in doses varying from 100,000 to 800,000 units in two to five days, alone or with the toxoid. Paralysis developed in 5 of the 3,481 cases of mild diphtheria, in 24 of 347 cases of severe diphtheria and in 38 of the 73 cases of malignant diphtheria. Fifty-five patients with malignant diphtheria died in the course of the first ten days of the disease. Paralysis developed more frequently in patients who had late or insufficient treatment than in those who had sufficient early treatment. It occurred on the ninth day of the disease in 1 case and between the thirteenth and forty-eighth day in 4 cases of moderately severe diphtheria, between the eighth and tenth day in 17 cases of severe and malignant diphtheria, and between the eleventh and fortieth day in 45 cases of severe and malignant diphtheria. The number of patients with paralysis totaled 101 and will be regarded here in four groups. The first and second group of 24 and 7 patients respectively were patients with severe or malignant diphtheria who received during the acute phase antiserum in doses ranging from 120,000 to 460,000 international units, alone or with toxoid. Local or more extensive paralysis developed between the eighth and the twentieth day in patients of the first group and completely regressed between the twentieth and the fifty-eighth day. Local or more extensive paralysis developed between the fifteenth and the forty-eighth day in patients of the second group. It regressed completely between the fortieth and the eightieth day after administration of the toxoid. The third group was made up of 15 patients with severe and malignant diphtheria who had no treatment during the acute phase of diphtheria or had late or insufficient treatment. Limited or extensive paralysis occurred between the sixth and the ninth day in 4 patients and between the thirteenth and the forty-ninth day in 11. Antiserum in a total dose varying between 100,000 and 500,000 international units and toxoid were administered. Regression with cure of paralysis took place within forty to eighty days in local or moderately pronounced paralysis and within seventy-five to one hundred and twenty days in some cases of extensive paralysis. The fourth group was made up of 55 patients with acute or malignant diphtheria. It included patients who had had no treatment in the acute phase of the disease, those who had insufficient or late treatment and those who had antiserum given in amounts from 200,000 to 500,000 international units during that phase of the disease. Paralysis occurred early in 10 patients and late in 45. The treatment consisted of administration of antiserum up to a total dose of 200,000 international units for patients not previously treated with antiserum, and of 100,000 international units for those who had antiserum in the acute phase of the disease. Recovery from paralysis took place between the twentieth and the hundred and twentieth day of the disease in all forms of local or multiple paralysis, which was controlled by repeated treatment if the condition reappeared after the first or second treatment. The author concludes that antiserum therapy in large doses administered early, in the course of diphtheria, alone or with toxoid diminishes the occurrence of diphtheritic paralysis. The repeated treatment controls recurrence of paralysis. The combined antiserum and toxoid treatment results in cure of limited and more extensive paralysis and of some but not all forms of multiple paralysis. The results of antiserum therapy alone are far superior to those of toxoid alone. Antiserum alone in primary or in repeated treatments produces a cure of diphtheritic paralysis of either limited or multiple types.

Chirurg, Berlin

12:253-284 (May 1) 1940

Question of "Permission for Operation." A. Hübner.—p. 253.
*Traumatic Pulmonary Collapse. L. Zukschwerdt and W. Pickel.—p. 258.
Spontaneous Hemorrhages of Spleen. J. X. Müller.—p. 265.
Malignant Carcinoids. F. Koch.—p. 270.
Cysts of Pavement Epithelium on Bones of Fingers. F. Becker.—p. 275.

Traumatic Pulmonary Collapse.—According to Zukschwerdt and Pickel, pulmonary collapse of traumatic origin has received little consideration. English observers called attention to it during the World War, particularly after gunshot wounds of the lung. The essential element in the traumatic pulmonary collapse is the deflation of a large portion of the lung. Authors' cases of traumatic collapse showed bronchial occlusion to be the cause of the deflation. Occlusion may be due to a thrombus caused by the pulmonary injury, and this may obstruct the bronchus of the injured portion of the lung, with a collapse of the injured side, or the thrombus may be transported, as the result of coughing, to the tracheal bifurcation and be aspirated into the opposite side, causing collapse of the noninjured side. The collapse may result from an injury to the bronchial mucosa with subsequent swelling and narrowing of the lumen, which later becomes completely blocked by mucus. The bronchus may be occluded by a plug of mucus. The gases in the portion of the lung distal to the occlusion

become absorbed. Suction toward the diseased part of the lung is transmitted to the surrounding organs. Elevation of the diaphragm, narrowing of the intercostal spaces, displacement of the mediastinal organs with the shift of the heart take place. Of the greatest functional significance is the displacement of the vessels at the base of the heart, because it interferes with their emptying. Added to this is the impairment of the circulation by excessive filling of the damaged lung with inadequately arterialized blood. The engorgement leads to circumscribed pulmonary edema, which may terminate in pneumonia or an abscess. If the causal factor is a thrombus the collapse may follow immediately on the injury, but if a plug of mucus is responsible several days may elapse before collapse takes place. The clinical signs are the same as in the postoperative collapse. Displacement of the heart toward the affected side (sign of Heller-Pasteur) is classic. An area of dulness with weakened respiratory sounds or with distant bronchial breathing and some crepitation are present in the beginning. Later the condition is that of a "dead lung." Roentgenoscopy is most reliable, because it discloses displacement of the heart and trachea. The temperature rises, the pulse becomes small and rapid, the face, at first brick red, soon becomes cyanotic. The expression is anxious, respiration superficial and accelerated. The course of traumatic pulmonary collapse varies. Bronchial occlusion may be cleared by coughing or by discharge of the secretion. Early death may be caused by mediastinal displacement. Many of the unexplained circulatory failures after chest injuries belong to the latter group. The persistence of the occlusion may lead to pneumonia, abscess, gangrene and empyema. Early treatment with substances which dissolve mucus is indicated. The authors used an aqueous solution of guaiacol or a solution of eucalyptus, menthol, acetanilid and phenyl-dimethyl pyrazolon in a nonacid oil, also inhalations. Hyperemia is produced by mustard plaster, by short waves or by turning the patient on the healthy side and back. Not too much time should be spent on these measures, especially if signs of circulatory collapse appear. If coughing does not clear the obstruction, a pneumothorax is indicated. The pneumothorax counteracts the mediastinal displacement. The circulation becomes normal and the suction on the occluding obstruction ceases, so that it can be expelled by coughing. Administration of remedies which dissolve the mucus should be continued. Adequate ventilation of the lung can be promoted by carbon dioxide. The authors are convinced that pneumothorax therapy is the method of choice. The pneumothorax should not be too extensive so as not to exclude too much of the healthy pulmonary tissue from respiration. The authors did not introduce more than 300 cc.

Deutsche medizinische Wochenschrift, Leipzig

66:1413-1440 (Dec. 20) 1940

Pneumonic Types as Determined by Reactive Status of Person. Frobenius.—p. 1416.

*Artificial Respiration and Resuscitation. R. Eisenmenger.—p. 1420.

Asphalt as Antigenic Agent. R. Hasche-Klünder.—p. 1422.

Artificial Respiration and Resuscitation.—According to Eisenmenger, experiments in artificial respiration and resuscitation conducted on recently dead bodies of animals and man demonstrated that the abdomen constitutes the only peripheral site at which respiration as well as circulation can be favorably influenced at the same time and that best results are obtained by apparatus mechanically controlling alternation of air pressure and air suction. The modern theory, revised from one held twenty years ago, is that what counts is not the amount of oxygen, expressed in cubic centimeters, passed through the lung but the simultaneous action on the heart and circulatory system by which the actual passage of the air to the capillaries of the lungs, heart and brain is assured in the shortest possible time and carbon dioxide eliminated. In serious cases of asphyxia in which respiration and circulation are involved, the latter constitutes the problem to be attacked first. It is with this end in view that intracardiac injections of epinephrine are made into the myocardium or right cardiac cavity to stimulate the heart and restore circulation. Direct cardiac massage in operative cases in which the heart is exteriorized and manipulated by the hand of the surgeon similarly is intended to incite the action of the heart. Both procedures need to be accompanied by artificial respiration. Biomotors are of two kinds, of over pressure and under pressure (überdruck and unterdruck). In the former an increased intrapulmonary pressure supervenes in the respiratory tract which affects the lesser circulation. In the latter, normal pressure relations prevail. By means of artificial pressure and suction apparatus in animal experimentation, epinephrine, caffeine and other drugs, intravenously injected, could be made to reach the heart and the central circulation even after the heart had already ceased its activity. Chemical agents can affect the nerve centers and thus influence blood circulation and distribution only if circulatory function still exists. If cardiac and circulatory action has already slowed down or ceased, mechanical intervention is in order. The author was able to observe the admirable results of mechanical resuscitation in cases of suffocation. Here cyanosis disappeared in from two to three minutes, consciousness was restored, acute liver enlargement receded and the patient could be promptly given nourishment. Artificial respiration by mechanical means may also be applied in serious cases in which sudden death appears to impend but no grave organic trauma is manifest and cardiac paralysis and acute circulatory insufficiency have not yet set in.

Klinische Wochenschrift, Berlin

19:409-432 (May 4) 1940

*Surgical Treatment of Hypertension. W. Nonnenbruch.—p. 409.

Aspects of Mediastinal Stasis. R. Schoen.—p. 413.

Anemias Resulting from Deficient Differentiation of Erythroblasts. R. Duesberg.—p. 417.

Dark Adaptation in Healthy Well Nourished Persons: Investigations on Range of Scattering with Adaptometer of Engelking-Hartung. R. Pies and H. Wendt.—p. 419.

Question of Surgical Treatment of Hepatocellular Icterus. W. Gros and W. Siede.—p. 420.

Casuistics of Acute Anterior Poliomyelitis. E. Altenburger.—p. 423.

Surgical Treatment of Hypertension.—Nonnenbruch advocates surgical treatment of hypertension for suitable cases. Kidney decapsulation and denervation of the renal hilus or combination of the two is one of the methods. Investigations suggest that the kidney influences blood pressure by humoral as well as by reflex action. The latter probably originates in the nerves of the capsule and in the pressoreceptor zones of the renal vessels. The second group of surgical interventions includes those on the sympathetic nervous system and on the adrenals, namely (1) unilateral and bilateral splanchnicotomy, with and without removal of the first and second lumbar ganglions and of the celiac ganglion; (2) removal of one or of a portion of both adrenals. Both groups of operations have also been combined in various ways. In the method of Adson and Craig, a subdiaphragmatic sympathectomy is combined with removal of the first and second lumbar ganglions and of a part of the celiac ganglion and with a partial excision of the adrenals. This method was employed also by Kirschner and by Schmieden. Analysis of cases of hypertension in which surgical treatment was employed reveals four different groups: (1) hypertension with true nephritis, (2) essential hypertension, (3) malignant nephroangiosclerosis and (4) blood pressure crises. The author emphasizes that an agreement must be reached as to what constitutes essential hypertension. The term should be applied only to cases with so-called benign sclerosis according to Volhard and Fahr, that is to cases of red hypertension, whereas cases of pale hypertension should be referred to as malignant sclerosis or as malignant hypertension. The two conditions differ. Red hypertension, as a rule, does not develop until the fifth or sixth decade of life and may persist for decades without impairment of the working capacity. In these cases surgical treatment is not necessary. However, in the early forms of malignant sclerosis and pale hypertension surgical treatment is advisable, for best results were obtained in such cases. They differ from benign sclerosis in that the patients are younger. Hypertension may be the only symptom for a long time, but the retinal vessels are as a rule considerably constricted. In some cases visual disturbances precede other symptoms by years, and an angiospastic retinitis or a cerebral attack caused by spastic ischemia is the first indication of the disorder. Urine and renal function may be entirely normal. According to Volhard pale hypertension is due to a hematogenically increased resistance in the arterioles, whereas red hypertension

is due to a decrease in the flexibility of the large arteries. The characteristic of the resistance hypertension is that the diastolic pressure is also high. In red hypertension, however, the systolic pressure is increased but the pressure falls considerably during diastole. In so-called malignant sclerosis there exist arteriosclerosis and a functional spasm. The latter may change in intensity, which explains a possible lack of stability of the blood pressure. The less fixed the hypertension, the more favorable the prospects of a successful operation. There are cases of pale hypertension in which the functional factor is primary. Such are cases of medullary tumors of the adrenals accompanied by hypertensive crises, also cases which begin with eclampsia. Cases with high pressure crises and normal pressure during intervals require careful observation. Surgical treatment is indicated in cases of paroxysmal hypertensive crises in which a paraganglion of the kidney is suspected, and also in early cases of malignant sclerosis in which clinical signs of renal disturbance are still absent. Surgical intervention to be considered first is renal decapsulation and interruption of the nerve trunks at the renal hilus. It is doubtful whether subdiaphragmatic sympathectomy with resection of the ganglions is justified. Further observations will be necessary to decide whether in cases of malignant sclerosis with permanent hypertension in which a paraganglion of the adrenal exists it is advisable to remove also one of the adrenals.

19:1321-1370 (Dec. 28) 1940. **Partial Index**

Number of Fluorescytes in Human and Animal Blood. E. Chytrek.—p. 1321.
Technic of Suboccipital Puncture for Functional Examination of Cerebrospinal Barrier. Vonkennel.—p. 1328.
*Comparative Explanation of Gastritis by Means of Gastroscopy and Stereoroentgenoscopy. L. von Friedrich.—p. 1333.

Gastroscopy and Relief Roentgenography in Gastritis.—Von Friedrich appraises the diagnostic value of the gastroscope in comparison with that of the mucosal relief roentgenograph on the basis of 273 cases of gastritis (atrophic gastritis 20, hypertrophic 140, ulcer complication 93). The mucosal relief roentgenographic examinations were simultaneously made by an independent investigator. While admitting the mutually complementary character of these diagnostic aids for gastritis, the author found gastroscopy superior, for though the roentgenograms report accurately the contours, movements and food passage of the stomach, they cannot determine the structure, color, nature and processes of the gastric mucosa. The atrophic type of gastritis, the nondiffuse, localized type (of greatest incidence), the hemorrhagic and erosive type, the postoperative type (counted among the most serious types of gastritis) and gastritis associated with ulcer all can be diagnosed accurately only by means of the gastroscope. What makes the explanation of gastritis difficult is the fact that the various types of gastritis have no specific causative agent nor do they present characteristic symptoms by which they might be identified. Unknown constitutional, endocrine and other factors affect the picture in each case. Repeated gastroscopic examinations of the same persons demonstrated not only the possibility of healing but the progress of the cure. The mere presence of coarse irregular folds and serrations in the greater curvature seen in stereoroentgenograms do not justify the diagnosis of gastritis or permit identification of the particular form of gastritis. The gastroscopic technic is simple, but the correct interpretation of the observations requires long experience.

Bulletin of the Naval Medical Association, Tokyo

29:833-894 (Dec.) 1940. **Partial Index**

*Two Cases of Osteomyelitis of the Frontal Bone. T. Masuoka.—p. 879.

Osteomyelitis of the Frontal Bone.—The incidence of osteomyelitis of cranial bones is rare, the statistical survey of 909 cases of osteomyelitis treated in the Tsukiji Charity Hospital, Tokyo, revealing only 0.22 per cent in the fifteen year period. The first patient seen by Masuoka was a woman 18 years old, who had a staphylococcic infection of the frontal bone following an injury to the forehead three years previous to admission to the hospital. Under local anesthesia the infected material was removed, together with two sequestrums embedded in granulomatous tissue; the patient went on to uneventful recovery. The second patient was a girl 4 years old, in whom frontal osteomyelitis followed abscess formations over the upper eyelids on both sides. The suppurative process extended into the intracranial tissues, causing an abscess of the frontal lobe of the brain and leptomeningitis of the base of the brain. The patient died. The author strongly recommends early and radical removal of the infected tissues, together with such chemotherapeutic measures as administration of sulfanilamide in full doses.

Klinicheskaya Meditsina, Moscow

18:1-136 (No. 10) 1940. **Partial Index**

Postoperative Massive Collapse of the Lung. K. A. Shchukarev.—p. 3.
Pathogenesis and Therapy of Hemoptysis. V. A. Chukanov.—p. 15.
*Problem of Grip in Pulmonary Tuberculosis. G. P. Rubinshteyn and F. M. Mandelshtamm.—p. 26.
*Liver and Bile Tract Involvement in Pneumonia. V. V. Gerbst.—p. 61.
Electrocardiograms in Lobar Pneumonia. I. S. Shnitser.—p. 69.

Grip and Pulmonary Tuberculosis.—Rubinshteyn and Mandelshtamm state that, of 648 patients admitted with the diagnosis of grip, roentgenologic studies revealed some tuberculous pulmonary lesion in 109, or 16.8 per cent. Of the 109, 21 presented single or multiple calcified foci or shrunken apexes with calcified foci and no signs of activity. These old lesions obviously had no relation to the attack of grip. Subtracted, this would leave 89, or 13 per cent. The authors made roentgenologic studies at periods varying from two months to three years after an attack of grip in 326 patients who, at the time of the grip attack, presented no roentgenologic signs of pulmonary tuberculosis. They failed to find a single case of pulmonary tuberculosis. Of the 109 grip patients with roentgenologic demonstration of pulmonary tuberculosis 18 presented infiltrative lesions, 71 inactive focal fibrous lesions and 20 fibrous cavernous process. In 71 (80 per cent) roentgenologic studies failed to reveal activation of the tuberculous process. Observations on 112 sanatorium patients with pulmonary tuberculosis revealed an incidence of 2.3 per cent of grip as compared with 10.3 per cent of incidence of grip in the hospital personnel. The authors conclude that grip apparently does not alter the reaction of the organism to tuberculous infection and does not favor the development of tuberculosis in areas previously free from tuberculosis on roentgenologic investigations. In the majority of the cases grip does not activate preexisting pulmonary tuberculosis. The authors have no data on which to base an opinion with regard to the effect of grip on the active fibrosing cavernous lesion. There are no characteristic symptoms capable of differentiating a flare-up of tuberculosis from that of grip. Tuberculous patients present a smaller incidence of grip and a milder course. The two infections are apparently not related to each other except for the similarity in the clinical picture.

Liver and Bile Tract Involvement in Pneumonia.—Gerbst studied 73 cases of lobar pneumonia and 27 cases of grippous bronchopneumonia with particular attention to the involvement of the liver and the bile tracts. He found that the abdominal syndrome complicating pneumonia manifests itself more clearly in the right upper quadrant than in the right lower quadrant. Pathologic alterations in the liver, gallbladder and bile tracts constitute one of the earliest and most frequent complications of pneumonia. Angiocholecystitis can be demonstrated clinically in 100 per cent of the cases with localization of the pneumonic process in the right lower lobe. With localization in the left lung the symptoms of liver-bile tract involvement were clearcut in 85.7 per cent, suggestive in 7 per cent and absent in 7 per cent. In grippous bronchopneumonia clinical symptoms of angiocholecystitis were present in 63 per cent. The pathologic alterations observed in six necropsies consisted of inflammation or of stasis of the gallbladder associated with dyskinesia of the bile tracts. The liver exhibited areas of focal hepatitis, necrosis, periangiocholitis and cholangitis. These alterations are, as a rule, reversible. The author believes that toxic factors of hematogenous character are responsible for these early lesions complicating inflammatory processes in the lung.

Book Notices

Report of the Blood Transfusion Association Concerning the Project for Supplying Blood Plasma to England, Which Has Been Carried on Jointly with the American Red Cross from August, 1940, to January, 1941. Narrative Account of Work and Medical Report. Paper. Pp. 121. New York: The Association, 1941.

In August 1940 the Blood Transfusion Association in New York was asked to aid in the development of plasma for shipment to England. This body was formerly known as the Blood Transfusion Betterment Association and first began its work in 1929. More recently it has been determined that all medical aid to Britain shall come through the American Red Cross. The record as here produced is an indication of an important activity and has value as a scientific medical document. Four thousand seven hundred and twelve liters was delivered for shipment to England; one shipment consisting of 222 liters was lost by the sinking of the S. S. *Western Prince*. The association not only aided in the collection of blood but provided funds for the study of new technics for drying plasma and has also developed a plan for collecting a hundred thousand different bloods. A bibliography is appended to this book.

Cancer and Occupation in Denmark 1935-1939. By Johannes Clemmesen, M.D. The Translation into English by Robert Fraser. Paper. Pp. 75. Copenhagen: NYT Nordisk Forlag-Arnold Busck, 1941.

This study is based on the official records of the Danish National Health Service, which are known to be well kept. It was supported by the Danish Anti-Cancer League, which has one hundred and fifty thousand members. As the population of Denmark is about 3½ millions, approximately one person in twenty-three is a member of the Anti-Cancer League. First come discussions of earlier Danish cancer statistics, the character of the Danish death certificates and the regional distribution of cancer in Denmark. The yearly deaths from cancer during 1935-1939 were 140 per hundred thousand of population. Then follow the classification of male "bread winners" in Denmark according to occupation with tables and analytic diagrams of the cancer mortality of the different occupation and age groups, general as well as according to sites. The outstanding result of the work "is the low mortality from cancer in agriculture and the high mortality from that cause in industry, in the age group 45-64," which, as the author puts it, "very strongly suggests that also in Denmark [as in certain other countries] occupation has a bearing on the development of the malignant growths." The significance of these results with respect to prevention of cancer is emphasized and the need of comprehensive statistical studies of cancer, morbidity as well as mortality, in relation to occupation is urged. The normal conditions in Denmark would be especially favorable for the establishment of such a center. "At a first glance it may perhaps seem that this will be a very expensive way of proceeding, but in practice it will be a far shorter way to knowledge about human cancer than the untold costly experiments on animals that are going on the world over, while we neglect the registration of the great, unhappy experiment which nature itself is carrying out on our fellow man."

Clinical Pellagra. By Seale Harris, M.D., Assisted by Seale Harris Jr., M.D. With foreword by E. V. McCollum, Ph.D., Sc.D., LL.D., Professor of Biochemistry, School of Hygiene and Public Health, the Johns Hopkins University, Baltimore. Cloth. Price, $7. Pp. 494, with 70 illustrations. St. Louis: C. V. Mosby Company, 1941.

The book is divided into an introductory chapter and seven sections dealing successively with history and epidemiology, the quest for the cause of pellagra, etiology and pathology, clinical investigations, symptomatology, diagnosis and prognosis, prophylaxis and treatment. These sections and chapters present a mixture of the good and the poor. For example, nowhere else can the student of pellagra find as complete a history of the disease, such a detailed yet clear description of the clinical pictures (well illustrated by photographs) found in pellagrins or the excellent summaries of recent clinical investigations into pellagra as conducted at Duke University and the University of Georgia. On the other hand, the chapter on the genesis of pellagra appears to have been written by one who resents the intrusion of nicotinic acid and other vitamins into a long held theory based on hypothetic toxins present in corn and alcohol. He states that "thus nicotinic acid deficiency in pellagra resulting from ingestion of toxins in corn products appears to be one of the many predisposing causes of the disease." Similarly the author attributes "alcoholic" pellagra partly to toxins present in the beverage and neglects entirely the fact that alcohol furnishes a vitamin free source of calories, which is sufficient to explain the examples chosen by the author to illustrate the toxic effects of alcohol in the production of pellagra. In the preface the author states "I have endeavored to write a factual treatise on pellagra in which the various phases of the subject are discussed, including summaries of the most important contributions by those who may be regarded as authorities on the subject." In general, the aim of the author to present a factual treatise on pellagra has been well done. It is when the author deviates from his purpose into a philosophic interpretation of the factual data that the book suffers.

Spermatozoa and Sterility: A Clinical Manual. By Abner I. Weisman, M.D., Clinical Assistant Visiting Gynecologist and Obstetrician, Metropolitan Hospital, New York. With a foreword by Robert L. Dickinson, M.D. Cloth. Price, $5.50. Pp. 314, with 77 illustrations. New York & London: Paul B. Hoeber, Inc., 1941.

Male sterility as a factor in childless marriages has been generally recognized only during the past twenty years. The knowledge of the spermatozoon has been meager, and textbooks of physiology contained little if any information. This book constitutes the first complete study of the subject. It fills, therefore, an urgent need. It covers the ground from the earliest history to latest advances, i. e. up to 1940, and is a valuable aid to every one interested in any of the phases of spermatozoal research. The practical application of the biologic, physical and chemical findings in the diagnosis and management of sterility in the male is extensively discussed and concise information is given, so that the book should be a great help for the practitioner working in this field. Chapters are devoted to the anatomy, physiology and chemistry of the spermatozoon, their metabolism and the composition of the seminal fluid. In others, causes of sterility in the male, methods for obtaining and transportation of specimen and evaluations are given. The influence of temperature, p_H and female genital secretions are discussed, among other subjects. The chapter concerning sterility produced by antibodies against spermatozoa is less critically reviewed than other subjects. The literature is partly misquoted, and conclusive experiments reported in 1940, excluding the possibility of temporary sterilization by spermatoxins, are not mentioned. The book is well bound and clearly printed.

Die Methoden der Fermentforschung. Unter Mitarbeit von Fachgenossen. Herausgegeben von Prof. Dr. Eugen Bamann und Prof. Dr. Karl Myrbäck. Lieferungen 2, 3 und 4. Paper. Price, 22.80 marks; 29.40 marks; 30.60 marks. Pp. 173-476, with 6 illustrations; 477-868, with 249 illustrations; 869-1276, with 177 illustrations. Leipzig: Georg Thieme, 1940.

The standard of thoroughness and clarity of presentation set by the first section of this reference work previously reviewed in THE JOURNAL has been well maintained if not surpassed in the present volumes. Section II continues the description of substrates, with papers on starch and glycogen and their split products, the uronic acids, the hexosephosphates, nucleic acids and polypeptides. The excellence of presentation is assured by the choice of the respective authors, e. g. Hirst and Peat on ascorbic acid, Abderhalden on polypeptides, and so on. Section III deals with the various methods used in the analysis of substrates, products and enzymes. These include, among others, roentgen ray analysis (Astbury) and the use of the polarigraph, ultracentrifuge methods (Lamm). Redox-potential measurements are described by Jörgen Lehmann. Chapters on the setting of the experimental conditions to be observed in enzyme work close this volume. Section IV continues with a detailed description of chemical methods for the analytic determination of a large group of organic substances as well as the use of colorimetry, spectroscopy and fluorescence measurements. Sufficient theoretical background for these methods is given to make this more than a mere practical manual. This volume also contains chapters on the use of manometric methods (by

Dickens) and the preparation of soluble enzymes from bacteria (by Werkman and Wood) as well as from plant and animal tissues. The text is printed in such a manner that quick reference is facilitated. However, all references to the literature are relegated to one volume, which will appear separately. The reviewer would have preferred to see the bibliography appended to the end of every chapter, even though this would have introduced a certain amount of duplication. At present, before all the sections of the handbook have been issued, reference to the original papers for further details is well nigh impossible. However, it can be said now that the work when completed will prove indispensable to all workers in biochemistry in view of the ever expanding interest in the field of biologic catalysts.

Medical Problems of Old Age. By Louis I. Dublin, Howard T. Karsner, O. H. Perry Pepper and Barney Brooks. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 46, with 6 illustrations. Philadelphia: University of Pennsylvania Press, 1941.

Chemotherapy. By E. K. Marshall Jr., John S. Lockwood and René J. Dubos. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 42, with 6 illustrations. Philadelphia: University of Pennsylvania Press, 1941

The Relation of Diseases in Lower Animals to Human Welfare. By John R. Mohler, Raymond A. Kelser and Cassius Way. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 39. Philadelphia: University of Pennsylvania Press, 1941.

Modern Aspects of the Antituberculosis Program. By J. Burns Amberson, Kendall Emerson, William Charles White and Louis I. Dublin. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 38. Philadelphia: University of Pennsylvania Press, 1941.

Female Sex Hormones. By Edward A. Doisy, Philip E. Smith, Robert T. Frank and Elmer L. Sevringhaus. University of Pennsylvania Bicentennial Conference. Paper Price, 50 cents. Pp. 58. Philadelphia: University of Pennsylvania Press, 1941.

Nutrition. By Conrad A. Elvehjem, Cyril N. H. Long and Elmer V. McCollum. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 46. Philadelphia: University of Pennsylvania Press, 1941.

Therapeutic Advances in Psychiatry. By Edward A. Strecker, Abraham A. Brill, Nolan D. C. Lewis and Arthur H. Ruggles. University of Pennsylvania Bicentennial Conference. Paper. Price, 50 cents. Pp. 35. Philadelphia: University of Pennsylvania Press, 1941.

Problems and Trends in Virus Research. By Thomas M. Rivers et al. University of Pennsylvania Bicentennial Conference. Paper. Price, 75 cents. Pp. 75, with one illustration. Philadelphia: University of Pennsylvania Press, 1941

From the University of Pennsylvania Press has come a series of booklets representing the lectures presented at the bicentennial conference. They represent an excellent inventory of our scientific knowledge in the field of medicine and health to which a number of distinguished investigators have contributed. The fields concerned are the medical problems of old age, chemotherapy, the relation of diseases in lower animals to human welfare, modern aspects of the antituberculosis program, female sex hormones, nutrition, therapeutic advances in psychiatry and problems and trends in virus research. The books are sold at most reasonable prices and offer opportunity to every reader to bring himself promptly abreast of current thought in the fields concerned.

The Chemical Action of Ultraviolet Rays. By Carleton Ellis and Alfred A. Wells. Revised and enlarged edition by Francis F. Heyroth, M.D., Ph.D. Cloth. Price, $12. Pp. 961, with 159 illustrations. New York: Reinhold Publishing Corporation, 1941.

The revision follows the same general plan of the original work published in 1925 but, as compared to the 362 pages in the first edition, comprises more than 950 pages. The text is not only a review of the mass of literature but also an effective analysis of the significance of the many contributions and is, as well, a complete manual for the investigator. The work would profit by the inclusion of more illustrative material in proportion to the profundity of the text. There is a disproportionate distribution of illustrations, e. g. there are four plates in the twenty page chapter on the commercial production of vitamin D but only ten in the preceding hundred pages dealing with the physiologic and therapeutic effects of ultraviolet rays. There is a somewhat better balance in the sections dealing with other industrial and commercial phases of the subject, which is a rich source of technical information that should be consulted by every one interested in the general problem of photochemistry.

Applied Physiology. By Samson Wright, M.D., F.R.C.P., John Astor Professor of Physiology, University of London, London. Seventh edition. Cloth. Price, $7. Pp. 787, with 366 illustrations. New York & London: Oxford University Press, 1940.

This is the seventh edition of a volume first produced in 1926. It is the twelfth printing. This in itself should indicate the quality of the work. The volume begins with two quotations which are well worthy of repetition as setting the key of the text:

> The simple believeth every word; but the prudent man looketh well to his going (Proverbs 14: 15).
>
> The student must continue to read good English and especially poetry, not only because the use of words is vital to the doctor but also that he may retain the flavor of a lovely language while he is studying from his textbooks (*Lancet*, Student Number, August 1939).

In his preface the author calls attention to the great accumulation of new information in the field of physiology. He points out that there have been over fourteen thousand abstracts of articles in this field published since 1939, and he hopes that he has been able to select the material of most permanent value for inclusion in his book. Indeed he submits in his preface page references to each of the places in which new information has been added. There are also one hundred and eighteen new illustrations.

Education for Family Life. Nineteenth Yearbook. Cloth. Price $2. Pp. 368, with illustrations. Washington, D. C.: American Association of School Administrators, 1941.

The American Association of School Administrators is studying the reasons for the breaking down of the home as the center of family life. This book is a report of the work of that organization. It supplies numerous recommendations that are helpful in this work, a bibliography, a report of the secretary and a list of the members.

Annual Review of Physiology. James Murray Luck, Editor. Victor E. Hall, Associate Editor. Volume III. Published by the American Physiological Society and Annual Reviews, Inc. Cloth. Price, $5. Pp. 784. Stanford University: Annual Reviews, Inc., 1941.

This volume is a compilation of the latest available physiologic data regarding many basic topics. The editors point out in the preface that they have been limited in their work by the failure of some of the reviewers to secure the most recent literature and by the failure of the editors to secure the cooperation of some colleagues who are engaged in war work. The volume offers a great deal of valuable information and is a most useful work of reference in the field of physiology.

Focus on Africa. By Richard Upjohn Light. Photographs by Mary Light. Foreword by Isaiah Bowman, President, The Johns Hopkins University, Baltimore. Cloth. Price, $3. Pp. 228, with 324 illustrations. New York: American Geographical Society, 1941.

Today Africa is the battleground of Europe's rivalries. Therefore every one has a far greater interest in that continent than previously prevailed. It is fortunate to have available this magnificent presentation of Africa by the American Geographical Society—a book based on airplane study with innumerable excellent photographs. Here are exceedingly valuable data concerning terrain and the population, and information regarding airports, maintenance and history of such developments as Uganda and Kenya settlements. There is a section on preservation of wild life and on the development of the radio. Finally there is an excellent bibliography and an index.

A History of Magic and Experimental Science. Volumes V and VI: The Sixteenth Century. By Lynn Thorndike, Professor of History, Columbia University, New York. Cloth. Price, $10, per set. Pp. 695; 766. New York, Morningside Heights: Columbia University Press, 1941.

These two volumes in this remarkable series deal with the sixteenth century. Here is a scientific account of the beginning of astrology, of the foundations of German medicine, the coming of Fracastoro, the rise of anatomy, of Jerome Cardan and Paracelsus. Here also is the story of the medicine of the period after 1550—superstitions relating to gems, divination and the literature of witchcraft and magic. This is certainly a most valuable contribution for all who are interested in the evolution of folk medicine and quackery.

Queries and Minor Notes

THE ANSWERS HERE PUBLISHED HAVE BEEN PREPARED BY COMPETENT AUTHORITIES. THEY DO NOT, HOWEVER, REPRESENT THE OPINIONS OF ANY OFFICIAL BODIES UNLESS SPECIFICALLY STATED IN THE REPLY. ANONYMOUS COMMUNICATIONS AND QUERIES ON POSTAL CARDS WILL NOT BE NOTICED. EVERY LETTER MUST CONTAIN THE WRITER'S NAME AND ADDRESS, BUT THESE WILL BE OMITTED ON REQUEST.

CHIGGER INFESTATION

To the Editor:—**I am an officer in the Army and would like information on the redbug, or chigger. This has been a serious problem, especially when we are out in the field. I should like to know (1) the life cycle of the insect, (2) method of entering the skin, (3) what happens to the insect after penetrating the skin, (4) what drug or drugs to use in prevention, (5) what treatment after the insect has caused the lesions and (6) does this insect attack other animals?**

M.D., Texas.

ANSWER.—1. The chigger is the six legged red larva of the division of Arthropoda, class of Arachnida, order of Acarida, section of Prostigmata, family of Trombidiidae. The eight legged adult is a scavenger and in the spring, after spending the winter underground, it deposits eggs on the ground and on low plants. In four weeks six legged larvae hatch. They are active and measure an average of 0.52 mm. in length and 0.31 mm. in width. Apparently these larvae must lead a parasitic existence in order to survive, and their hosts are reported to include domestic animals, rodents, birds, certain reptiles and man. The larva does not change hosts; it rarely remains attached longer than two to four days and then drops off, engorged with a fatty predigested tissue juice (not blood). Then it goes into a pupal form, and in a week or two a colorless, eyeless eight legged nymph emerges. After a few more days a second nymph form appears, and after two or three more weeks the nymph becomes an adult. The nymphs and adults live on plants and decayed vegetation. They are scavengers and do not parasitize man or animals. There is only one generation of chiggers each year.

2. When a person walks through grass or underbrush he may become infested as he comes in contact with vegetation on which the chiggers are presumably located. Perhaps they may also be found on the leaves of small trees. They may invade the skin of the host from the feet and ankles upward or, less commonly, from the neck and shoulders downward. They run at the rate of about 10 cm. per minute and are most likely to attach themselves at points where they meet an obstacle, such as a constricting garter or belt. They prefer areas where the epidermis is thin. The bite is usually, but not always, at the mouth of a follicle, the mite being fastened temporarily as a red speck at the center. The larva does not burrow but remains fastened on the surface, like a tick, merely piercing the epidermis as deeply as possible with its pointed mandibular claws. The symptoms are probably caused by the digestive secretion of the mite, which flows into the wound. A pruritic papule or papulovesicle appears.

3. The mite becomes engorged and usually drops off after a few hours and can rarely be found unless the patient is seen early.

4. Infestation may be prevented by dusting the skin with sulfur or by wearing protective clothing.

5. There are three objectives in treatment: (1) the removal of the mites, (2) the relief of the severe itching and (3) the treatment or prevention of secondary infection. To remove the mites, if still present, kerosene may be applied, followed by a half hour of bathing with plenty of soap and a complete change of clothing. Active mites may remain in infested clothing. Since the objective and subjective symptoms usually continue unabated for days after the parasite has been removed, the fulfilment of the second and third objectives is of the greatest importance and may best be accomplished by touching the lesions with 70 per cent alcohol (rubbing alcohol) three times a day and as needed, followed immediately by a mild antiseptic antipruritic ointment, such as boric acid ointment U. S. P., to which may be added from 1 to 2 per cent of phenol, the strength of course being in inverse proportion to the area of skin to be covered, and 0.2 per cent of menthol. This ointment should be applied sparingly at least three times a day (after alcohol) and used also as needed to relieve itching; it is to be rubbed in gently, and the remainder is wiped off with cotton. A little plain talcum may then be dusted over the surface. Scratching must be prevented, and canvas gloves may have to be worn during sleep for this purpose. If serious secondary infection has occurred, of course other measures may be used as indicated.

6. This mite is a larval acarid and not an insect. It attacks sheep, goats, cattle, dogs, house cats, monkeys, rabbits, squirrels, moles, rats, house and field mice, bats, hedgehogs, shrews, voles, opossums, tinemous, domestic fowl and certain ground nesting birds, frogs, toads, salamanders, lizards, certain snakes, land turtles, insects and arachnids.

References:

Toomey, Noxon: Trombidiasis (Leptus Autumnalis), *Urol. & Cutan. Rev.* **25:** 598 (Oct.) 1921.

Parkhurst, Howard J.: Trombidiosis (Infestation with Chiggers), *Arch. Dermat. & Syph.* **35:** 1011 (June) 1937.

HIGH TEMPERATURES AND HEMIPLEGIA

To the Editor:—**A man aged 59 has had cardiorenal hypertension for many years. He has worked for twenty years stoking a furnace. Four months ago he worked all day as usual and felt well, complaining however of the heat more than usual. He walked home 2 miles as was his custom. Four hours after his evening meal he was stricken with a hemiplegia. The man is claiming that the hemiplegia was caused by the heat and his occupation as stoker. What are the possibilities of this being the case? How long after being overheated do symptoms of heat stroke appear? Will you also give me some references regarding heat stroke causing hemiplegia?**

Philip C. Thomas, M.D., Knoxville, Tenn.

ANSWER.—It is first necessary to distinguish between a heat stroke and a stroke in the common sense, such as a cerebral hemorrhage, which would cause hemiplegia. Exposure to high temperatures, especially associated with high humidity, may lead to a train of symptoms varying from cramps to sudden death. If the condition is merely one of heat exhaustion the mouth temperature is usually normal but sometimes may be persistent if the exposure to heat continues, as in the tropics.

Heat stroke or sun stroke is accompanied by a marked elevation of temperature, from 106 to 109 F. This may come on suddenly, and consciousness is usually lost early in the attack. If the patient survives for several days, recovery is usually complete; but susceptibility to heat and impairment of memory are common results.

It is obvious in the face of these facts that a patient 59 years old who has had hypertension for many years and has worked for twenty years at the same employment of stoking a furnace, even though this would expose him to heat, could not attribute a hemiplegia coming on four hours later to his occupation; at least it is unlikely that this contention is probable, and it is quite unlikely that the two are associated.

References:

Glover, D. M.: Heat Cramps in Industry, *J. Indust. Hyg.* **13:** 347 (Dec.) 1931 (bibliography).

Morton, T. C. St. C.: Etiology and Treatment of Heat Exhaustion and Heat Hyperpyrexia, Proc. Roy. Soc. Med. **25:** 261 (June) 1932.

Sayers, R. R., and Davenport, S. J.: Review of Literature on the Physiological Effects of Abnormal Temperatures and Humidities, Pub. Health Rep. **42:** 933 (April 8) 1927; Reprint 1150 United States Treasury Department, Public Health Service, 1927 (extensive bibliography).

UNFAVORABLE REACTION TO ORANGE AND TOMATO JUICES IN INFANT

To the Editor:—**There is much in the literature about the use of vitamin D in massive doses in the treatment of arthritis. More recently articles have appeared on the use of vitamin B for the same purpose. I have heard arguments in favor of each. Will you please tell me which is the more useful? I am caring for a boy who has had much trouble with gas from the time he was 6 weeks old. However, at that time his parents had him on a trip, and his trouble was attributed to that for a while. After he was home and settled down, it was discovered that he cried and had pain from gas if he had orange or tomato juice in the morning, but his trouble did not begin until after 6 p. m. and would bother him most of the evening. If he expelled gas, he was relieved. An enema would relieve him. After the elimination of these juices from his diet, he had no trouble. If his diaper is not changed and his skin washed well after every stool, his buttocks become as red as if they had been scalded. He is now 9 months old and cannot take tomatoes without irritation of his buttocks; even ascorbic acid will give the same reaction. He had no form of vitamin C yesterday, but his diaper was not changed immediately after a stool in the night and today his buttocks are greatly irritated. Why is this, and what method can be used to supply vitamin C?**

M.D., Nebraska.

ANSWER.—In spite of the fact that a considerable number of papers have been published on the subject of treatment of arthritis with massive doses of vitamin D, it must be recognized that this form of therapy is still in the experimental stage. Negative as well as positive results have been reported. Nothing is known of the physiologic or pharmacologic mechanism by which this vitamin might effect a cure of arthritis. The dose used by

various clinical investigators has varied between 150,000 and 1,000,000 international units of vitamin D a day. Since there is more convincing evidence of the harmful effects from this large dose of the vitamin when used over a considerable period, the method cannot be recommended for regular use until its value has been more firmly established.

While the value of certain factors of the vitamin B complex, particularly thiamine hydrochloride, in the treatment of certain forms of neuritis has been thoroughly proved, evidence of any specific value in arthritis is inadequate at the present time. The most that one could say for it with certainty is that the metabolism of the body as a whole is safeguarded by including adequate amounts of all the vitamins in the daily diet.

It is possible that the infant described is hypersensitive to the proteins in tomato or orange juice, although this is an unusual occurrence. Since all infants require vitamin C, this must be given. If he persists in showing the undesirable effects referred to from taking these juices, it would be necessary to give vitamin C in the form of ascorbic acid. The daily requirement for a baby aged 9 months is somewhere between 25 and 50 mg., an amount which could be dissolved in the formula. Attempts to give orange or tomato juice in gradually increasing amounts should be made as the baby grows older. It is unlikely that these juices are the cause of the intestinal gases referred to. The latter are more likely due to the swallowing of air when the baby is taking his feeding.

ACUTE SUBDELTOID BURSITIS OF PERIARTICULAR FIBROSITIS: PAINFUL SHOULDER

To the Editor:—**A woman aged 21, while in Florida, suddenly complained of severe stiffness and pain in the left shoulder. She consulted a physician, who made a roentgenologic study of the shoulder but found nothing wrong. The patient was apparently in excellent health. There was inability to elevate or rotate the shoulder without excruciating pain, and tenderness over the coracoclavicular articulation. There were some pain and tenderness over the lower cervical and lumbar spine on attempted motion and palpation. There was some pain on flexion and extension of the phalangeal articulations in both hands. The blood pressure was 112 systolic and 74 diastolic, the pulse rate was 72; temperature was 98.6 F., respiratory rate 18. The sedimentation test was 7 mm. per hour, red count 4,900,000, hemoglobin 90 per cent, white count 7,500, differential normal, Kahn reaction negative. Cervical smears and gonococcus fixation tests were negative. Urinalysis was negative. A roentgenogram of the teeth was negative and of the hands negative for disease of bone. There was a gingival abscess in the region of the third molar which was opened and drained ten days prior to her visit to Florida. She is an excellent swimmer, giving numerous exhibitions weekly, and is active athletically. She has improved markedly on salicylate medication orally and intravenously, diathermy, and active and passive movements. There is, however, some residual stiffness on arising in the morning. Cold and damp weather also aggravates her condition. Would this be in line with a diagnosis of acute periarticular fibrositis? Should she give up swimming? What is the prognosis, and what further treatment might be instituted?**

M.D., Chicago.

ANSWER.—The absence of fever, with negative roentgenograms of the shoulder and normal values for the sedimentation test and the leukocytes, indicates that the condition does not represent infectious arthritis of the shoulder. Most likely the patient had what is generally called "acute subacromial or subdeltoid bursitis" or "humeroscapular periarthritis" associated with muscle spasm and inflammation of the adjacent muscles and tendons. Such an event is a common occurrence among athletes, especially among those who use their arms vigorously. Among conductors of orchestras, violinists or baseball pitchers the condition is called "glass arm," "musician's shoulder" and the like. As the patient is an active swimmer and presumably did considerable swimming during her recent vacation, she may easily have injured the left shoulder without being aware of it.

According to Codman (The Shoulder: Rupture of the Supraspinatus Tendon and Other Lesions In or About the Subacromial Bursa, Boston, Thomas Todd Company) and many others the lesion of subdeltoid bursitis generally is not in the bursa but in the tendon of one of the rotators of the arm, especially in the supraspinatus tendon. Minor but repeated injuries to this tendon may produce small tears with microscopic hemorrhage or inflammation, leading to small patches of scar tissue which further weaken the tendon. Later an insignificant injury may precipitate an extremely acute and presumably spontaneous inflammation in the tendon. In some cases the adjacent tissues become involved, and inflammation and muscle spasm affect the articular capsule and surrounding muscles. When this ("acute capsular rheumatism" or "acute periarticular fibrositis") occurs, the patient's pain often forces him to keep the arm fixed with the shoulder and arm sharply adducted. Unless the shoulder is treated properly and placed in proper abduction, it may become fixed in adduction with permanent partial loss of abductors and posterior rotators.

The role of infection in such cases has not been proved. It would be well, of course, to remove obviously infected foci. The fact that the patient had slight soreness in regions other than the shoulder indicates, perhaps, the presence of mild disseminated fibrositis.

For the time being the patient might use the shoulder within the limits of its proved tolerance, that is, mild active and passive motions of the shoulder are to be desired in conjunction with physical therapy and mild daily manipulations. Vigorous use of the arm, however, such as necessitated by swimming, should be avoided temporarily and not indulged in until ordinary use is essentially painless. Many patients note that, when the acute process subsides, residual soreness can be ameliorated by lying at night with the arm over the head or by sleeping with the head on two pillows and another pillow under the affected arm. Later, perhaps, the patient might temporarily alter her swimming stroke and use one which would traumatize the affected shoulder less than the crawl or other strokes. The arm and shoulder may recover completely, but in many cases there is a tendency for the recurrences of this condition from time to time.

DIABETES AND ALCOHOL

To the Editor:—**I would appreciate any information or references you could send me concerning the ingestion of alcohol in diabetic patients: the caloric value, the effect of it on insulin intake and metabolism in these patients.**

Leo Keller, M.D., New York.

ANSWER.—The caloric value of alcohol is 7.1 calories per gram as compared with carbohydrate 4.1, protein 4.1 and fat 9.3 calories. Since it contains no carbohydrate, it should have no influence on the need for insulin unless by its use the total caloric value of the diet is in excess, in which case more insulin should be required, in conformation with the original investigation of Allen and Wishart (Alcohol in the Diabetic Diet, *J. Metab. Research* **1**:304, 1922). They concluded that "alcohol, which is clearly recognized as not convertible into sugar or acetone in the body, produces a return of glycosuria and other symptoms when added to the diabetic diet in quantities exceeding caloric tolerance." Obviously, if alcohol is used in calorific value to replace carbohydrate, the requirement for insulin should fall.

Alcohol in equivalent dosage should have the same effect on the metabolism of a diabetic as on a normal individual. It is devoid of vitamins. It does not increase or decrease acidosis.

References substantiating these statements about the use of alcohol in diabetes by Newman and Cutting, by Higgins, Peabody and Fitz, by Allen and Wishart, by Leclercq, by Fuller, by Strouse, Soskin and Vidgoff and a discussion of the value of alcohol in diabetes are given in the seventh edition of the Treatment of Diabetes Mellitus by Joslin, Root, White and Marble (Philadelphia, Lea & Febiger, 1940). In Wilder's monograph (Clinical Diabetes Mellitus and Hyperinsulinism, Philadelphia, W. B. Saunders Company, 1940, pp. 159-160) he refers to his original article in Emerson's "Alcohol and Man," 1932.

The whole question of insulin and its effect on the metabolism of alcohol has been reviewed by Thorne M. Carpenter, acting director, Nutrition Laboratory, Carnegie Institution of Washington, and published in a paper entitled The Metabolism of Alcohol: A Review, with many references (*Quart. J. Stud. Alcohol* **1**:201 [Sept.] 1940). A concluding sentence is "In general, when insulin is given in large doses, some increase in the rate of disappearance of alcohol takes place, but this hormone is more effective when combined with glucose."

For the use of insulin in alcoholism, apart from diabetes articles have recently appeared by Taylor and Cross (Treatment of Acute Alcoholism with Insulin, *J. Pediat.* **16**:341 [Feb.] 1940, Wortis, Bowman and Goldfarb (Use of Insulin in Alcoholism, *M. Clin. North America* **24**:671 [May] 1940) and Goldfarb, Bowman and Parker (Glucose and Insulin in Acute Alcoholism, *J. Clin. Investigation* **18**:581 [Sept.] 1939).

In Plato's ideal state, the diabetic might profit by the use of alcohol, but in the United States, where so many exist and are trying to earn a living, the diabetic who drink are a menace to themselves and to others, because, although they may not be intoxicated, an alcoholic breath may obscure a hypoglycemic reaction and bring those who have it and at the same time other diabetic persons into disrepute. The diabetic are as liable, and perhaps more so than other persons, to complica-

tions of alcoholism, such as neuritis and amblyopia, the latter condition also having tobacco as an accessory.

Wilder states that "the use of alcohol in diabetes is no longer an important problem. That it has some antidiabetic effect has been shown, but this is too feeble compared with that of insulin to be of practical significance. . . . On the other hand, there is no contraindication in diabetes to the consumption of moderate quantities of alcoholic beverages."

The first sentence of the directions for diabetic patients at the Beth Israel Hospital in Boston is as follows: "Drink no tonics, no wines or other liquors," and we do not recall any hospital which advises its diabetic outpatients to use alcohol in any form.

Joslin tells his patients to leave alcohol alone.

SPASTIC PARAPLEGIA IN FLEXION

To the Editor:—A man aged 36 has been confined to bed for one and one-half years with multiple sclerosis. About three months ago, a flexor contraction of the legs developed which is so intense that the legs are almost completely flexed on the abdomen. Occasionally a paroxysm of flexion will attempt to increase this already aggravated condition. It is impossible to straighten out the legs. The position is painful and makes nursing him a great chore. I read an article in the *Saturday Evening Post* in November 1940 in which was a description of how curare had been used on persons with spasm. Have you any information regarding the use of curare in these extreme contractions? A neurologist suggests chordotomy for the relief of this patient, but if such measures are contemplated I think that even the unknown risk of using a drug like curare might be justifiable. Any suggestions or help will be greatly appreciated.

M.D., Pennsylvania.

ANSWER.—This patient apparently has a clinical condition known as spastic paraplegia in flexion. This always indicates diffuse disease of the spinal cord. He has also spontaneous contractions of the muscles of the legs due to diffuse disease of the spinal cord with almost complete transection. Curare has been used lately for patients suffering from generalized rigidity and increased motion (tremor) due usually to cerebral disease. It also has been used before injections of metrazol for shock therapy in psychosis. It is suggested that the patient be hospitalized and an attempt be made by means of absolute rest and weight pulling on the legs either to diminish or to remove the spasticity. Passive motion, massage and hydrotherapy in the form of whirlpool baths may help. Scopolamine hydrobromide may be given. If after several weeks no improvement takes place, curare may be tried or a section of the posterior roots on both sides in the lower dorsal and upper lumbar regions can be done. Chordotomy per se is done only for the relief of pain. If all the anterior horns in the involved area are sectioned on the two sides then one may have a reduction of the spasticity. This latter operation is not chordotomy and is not recommended. Neither is chordotomy. One must be certain that one is not dealing with a spinal block, which would mean a tumor of the cord. If spinal manometric study has not been done it is recommended that it be done before such procedures are carried out. Patients with spastic paraplegia not due to a tumor of the spinal cord or subacute combined degeneration of the cord offer considerable difficulty in management.

METASTASES FROM CARCINOMA OF ESOPHAGUS

To the Editor:—Will you explain metastasis from the lower part of the esophagus to Virchow's gland in carcinoma of the esophagus? I have a case in which the esophageal lesion showed only hypertrophied rugae in the lower end without any destruction of the surface epithelium. At biopsy this tissue was diagnosed as carcinomatous, and the enlarged gland over the inner third of the left clavicle was said to be metastatic.

E. W. Carpenter, M.D., Greenville, S. C.

ANSWER.—In esophageal carcinoma, metastasis may take place by way of lymph and blood vessels. All the coats of the esophagus have networks of lymph vessels which pass to the lower deep cervical and to the posterior mediastinal lymph nodes. From primary carcinoma in the esophagus secondary growths may arise in the neck, lungs, mediastinum, liver and other places. Even extensive involvement of cervical nodes may take place in small cancers of the esophagus with indefinite or no symptoms. "Virchow's gland" is the lymph node or nodes behind the insertion of the left sternomastoid muscle near the termination of the thoracic duct at the junction of the left subclavian and internal jugular veins. In 1849 and also earlier Virchow pointed out that in carcinoma of the abdomen secondary growths may develop in these nodes, a localization that might be of diagnostic value. The present case illustrates that in cases of enlargement of "Virchow's gland" the possibility of a primary carcinoma of the esophagus should not be neglected.

ACNEFORM ERUPTION IN GASOLINE STATION ATTENDANT

To the Editor:—May I ask your opinion with regard to a patient who works as a gasoline station attendant and is troubled with a cutaneous eruption of a papular acneform nature on his scalp and over the anterior aspect of both thighs. He adds that the papules rarely, if ever, go on to the formation of pustules.

M.D., Massachusetts.

ANSWER.—Papular acneform eruptions on the anterior aspect of the thighs are of frequent occurrence among workers whose trousers become soiled with petroleum and coal tar products, such as oils and greases (Schwartz, Louis, and Tulipan, Louis: A Textbook of Occupational Diseases of the Skin, Philadelphia, Lea & Febiger, 1939, pp. 190 and 616). Daily change to clean underclothes and work clothes, together with cleansing baths after work will prevent the occurrence of this condition. While no cases of such a papular acneform eruption of the scalp caused by petroleum or coal tar products have been reported, it is a possibility that grease on the scalp may produce this condition. However, it seems more likely that the cutaneous eruption on the scalp is due to some other cause.

HIGH FAT DIET IN CHRONIC CHOLECYSTITIS

To the Editor:—A patient with chronic cholecystitis obtains relief from daily and frequent ingestion of eggs and cream. Would this be contraindicated, having in mind the danger of gallstone formation? Cholecystography is negative for calculi. The blood cholesterol is normal.

M.D., New York.

ANSWER.—There is no completely satisfactory clinical or experimental evidence to indicate that a high fat, high cholesterol intake of itself will increase the amount of cholesterol in bile or favor the development of gallstones. Such a diet may be of more etiologic significance if infection or biliary stasis is present. The patient in question may have some functional type of biliary stasis which produces distress; under such circumstances the diet mentioned, which should produce a maximal degree of motor activity, is desirable. High fat intakes and the use of bile acids and antispasmodics have been recommended even in the presence of gallstones (Mock, H. E.; Brown, C. F. G., and Dolkart, R. E.: The Conservative Treatment of Gallbladder Disease, *Surg., Gynec. & Obst.* **66**:79 [Jan.] 1938.) and seem to produce symptomatic improvement in many cases.

ACCELERATED REACTION TO SMALLPOX VACCINATION

To the Editor:—In June 1940 I vaccinated a boy aged 8 years who had previously been vaccinated twice with reported failure. When I vaccinated him, the typical red area, vesicle and brownish scab developed. It was, however, an accelerated reaction, being completed in about seven or eight days, and there was little foveation or puckering or scar through the epidermis down to the corium. I gave him a certificate of vaccination. I have just learned that the school physician refused to honor the certificate because he says there is insufficient scar. (I might also add that this vaccination was done under aseptic conditions with an approved fresh virus.) It is my opinion that, if one has a high immunity, revaccination will often give an accelerated reaction and leave little or no scar. Is this assumption correct? Do you believe this boy should be vaccinated again and again until he gets a sizeable scar? If my knowledge of immunity is correct, he will get less and less of a scar (unless there is secondary infection) when he is revaccinated time and again.

M.D., Pennsylvania.

ANSWER.—The accelerated reaction described is valid evidence of protection against smallpox. There is no reason whatever for further vaccinations at this time or for refusal to honor the certificate of successful vaccination.

BLOOD TYPE DOES NOT CHANGE

To the Editor:—I should like to add a few remarks to the answer to the query "Blood Type Does Not Change" in The Journal of June 28, page 2893. The query referred to two questions; (a) the apparent change in the blood group from O to A and (b) the absence of a reaction following the transfusion of blood of group A to a recipient whose blood was not stated. The first question was answered adequately. One could add to the answer to the second question two remarks. The low titer of isoagglutinins in the recipient may explain the absence of a reaction when incompatible blood is transfused. Such low titers occur not infrequently. They are especially common in the aged and in patients with chronic leukemia. The effect of a low titer of isoagglutinins in the recipient would be especially noticeable in a case of a donor of subgroup A_2, which is characterized by a low agglutinability. Another consideration in this case has to do with the absence in the query of a statement regarding the recipient's blood group. The donor was originally thought to belong to group O, which group is used for recipients of all blood groups. If the recipient in this case was of group A, the then absence of a reaction would be expected.

Israel Davidsohn, M.D., Chicago.